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PHYTOPATHOLOGY

VOLUME X NUMBER 11

NOVEMBER, 1920

COLLAR-ROT OF APPLE TREES IN THE YAKIMA VALLEY

J. W. HOTSON

WITH 15 FIGURES IN THE TEXT

INTRODUCTION

The Yakima Valley is one of the principal fruit-growing regions of the State of Washington. During the years 1913–1916, fire-blight became so prevalent that it threatened the very existence of the apple industry in the Valley. By drastic action this disease, although not exterminated, is being partially controlled. At the present time (1920) the orchardist is facing another type of trouble that, largely on account of the subtleness of its behavior, promises in the long run to be almost, if not quite, as serious a menace as the fire-blight. A general survey of the orchards from Selah to Grandview revealed the prevalence and seriousness of diseases attacking fruit trees at or near the crown and generally spoken of as "collar-rot" or "crown-rot."

The present article has to do with this malady as it occurs on fruit trees in the Yakima Valley, where most of the investigation was carried on, although some experiments were made on trees in the vicinity of Seattle.

LITERATURE

The literature on this subject has been quite fully reviewed by Grossenbacher (18).[1] That particular phase of the subject relating to arsenical poisoning is also reviewed by Swingle and Morris (37). Various writers working on similar types of injury have attributed the trouble to a variety of causes. In Illinois (4), New York (18, 35), Wyoming (25), Nebraska (11–12), and Connecticut (6) it has been reported to be due to winter injury or frost, while in West Virginia (16), Pennsylvania (26), and certain parts of New York (38) it has been definitely associated with the fire-blight

[1] Reference is made by number to "Literature cited," p. 484.

organism, *Bacillus amylovorus*. In Colorado it has been attributed to excessive irrigation (28), woolly aphis (28), and arsenical poisoning (23 24, 39). *Clitocybe parasitica* has been shown to be the causal organism in Oklahoma (40), *Xylaria polymorpha* in Virginia (13, 14, 15) and North Carolina (41), while *Armillaria mellea* has been found associated with a similar condition in various parts of the United States. Other fungi, like *Polystictus versicolor*, *Thelephora galactina* (31), *Sphaeropsis* (18, 27), and *Pleurotus* (18), have been occasionally found associated with collar-rot cankers.

FIG. 1. AN ORCHARD SHOWING APPLE TREES IN THE ADVANCED STAGES OF COLLAR-ROT

Those in the foreground are already dead

EXTENT AND DISTRIBUTION

If collar-rot means the presence of a canker at or near the crown of a fruit tree, from whatever cause, then it would appear from the above that this disease is quite widely distributed over the United States. In the Yakima Valley it is considered extremely serious. The examination of a certain 5-acre orchard near the city of Yakima showed twenty-five trees in which the disease could be readily detected simply by the foliage. By a careful tree-to-tree inspection, removing the soil from the base of each tree to a sufficient depth to determine whether it was infected, over one hundred trees were found showing various stages of development of the disease. An orchard more seriously affected than the one just mentioned

is shown in figure 1, where, from the point from which the photograph was taken, at least thirty affected trees could be recognized, those in the foreground having already died. An examination of other orchards

FIG. 2. A TREE AFFECTED WITH COLLAR-ROT

Showing a general sickly appearance, with small leaves sparingly developed

emphasized the fact that, although only a few trees might show the effect of the disease on the foliage, a much larger number showing the canker without the foliage symptoms could invariably be found by making a careful survey.

SYMPTOMS

The most striking symptoms of collar-rot are seen during the growing season. The leaves become yellowish and much smaller than normal ones. In the earlier stages of discoloration of the foliage, a single limb or portion of it may show signs of the disease. This limb usually appears on that side of the tree on which the canker occurs; at least it lies along that portion of the vascular system that passes through the cankered area. As the disease progresses the whole tree takes on a sickly appearance, and the limbs, especially on the south side, become a light yellowish or yellowish-red color and more or less scalded in appearance. This condition of the bark may be due in part to malnutrition, but it is mainly due to the excessive action of the sun, since there is only a limited amount of foliage.

FIG. 3. A TYPE OF COLLAR-ROT WHERE THE INITIAL INJURY WAS AT THE CAMBIUM OR PHLOEM

These general symptoms are sufficient for the detection of a tree in the advanced stages of the disease nearly as far as it can be seen. One of these trees is shown in figure 2, and one of the cankers in figure 3.

An examination of the crown of such a tree reveals a more or less conspicuous sunken area. This canker varies in size according to the length of time the disease has been working and the character of the original wound. As the bark is killed it appears brownish, and in advanced stages frequently becomes almost completely transformed into a chocolate-colored powder. In some instances the outer portion of the bark is left intact, while the inner part becomes more or less disintegrated. The disease gradually extends downward into the roots, less rapidly upward in the trunk, and very slowly laterally. The wood beneath the affected bark often becomes water-soaked, brownish in color, and frequently charred in appearance. This discoloration eventually extends into the heart of the stem and root.

One of the serious features of this disease is the fact that, since the trouble begins under ground or near the surface, it is overlooked by the orchardist until some evidence is seen in the foliage, the whole tree taking on a sickly appearance. Unfortunately, at this stage the disease has frequently become so far advanced that no effective remedy can be applied.

ORGANISMS ASSOCIATED WITH COLLAR-ROT

When a wound fails to heal an opportunity is given for a variety of organisms, either parasitic or saprophytic, to become established and eventually to gain entrance to the center of the tree. In the Yakima Valley definite organisms, such as *Armillaria mellea*, *Polystictus versicolor* and *Bacillus amylovorus*, have been found associated with these cankers, *B. amylovorus* having been shown to be the primary cause of some of them. *A. mellea* was found definitely associated with at least twenty different apple trees, and reports were received of its occurrence on many others. In some instances the rhizomorphs had but recently invaded wounds of long standing, while in other cases they seemed to have become established shortly after the wound was formed, and in still others they almost completely covered the roots without any apparent infection. This fungus was identified by the typical, shining, grayish-black rhizomorphs found mostly just below the bark (fig. 5).

It is recognized that the presence of these rhizomorphs is not an absolute proof that the organism is *A. mellea*, but a comparison of the cultural characters of some of these grown on potato agar with those of a known pure culture leaves little doubt that the rhizomorphs found in connection with diseased trees belonged to this organism. In a few instances after the trees had died sporophores were found, but in the majority of cases, the only means of identification was the rhizomorphs.

Polystictus versicolor was found occasionally on apple and prune trees, especially in old orchards. It is not limited to its attack on the roots, but occasionally, like *A. mellea*, it may gain entrance to the tree at a wound near the crown, and from this infected area the mycelium may spread upwards and downwards, producing heart-rot. A prune tree attacked by *P. versicolor*, which is producing sporophores, is shown in figure 7. This tree was first observed in the spring of 1914 with its bark peeled off just at the ground, leaving an irregular wound about the size of one's hand. A superficial examination showed no fungus although the tree may have been infected at that time. During the summer of 1915 the tree began to show signs of root trouble, in the leaves, and by middle of August it was dead. That fall sporophores began to form, and in the following spring when this photograph was taken they were abundant.

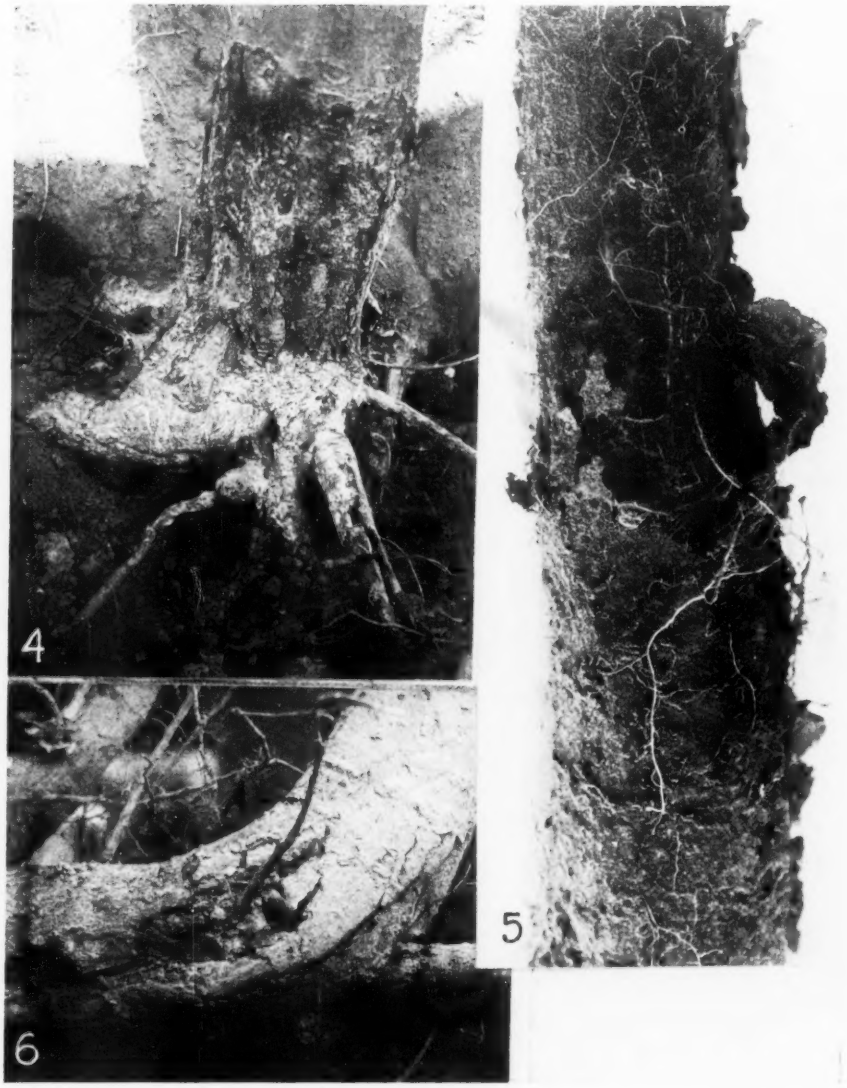

FIG. 4. A TYPE OF COLLAR-ROT PRODUCED BY A PROCESS OF CORROSION FROM THE OUTER SURFACE OF THE BARK

FIG. 5. RHIZOMORPHS OF ARMILLARIA MELLEA ON THE ROOT OF AN APPLE TREE

FIG. 6. A YOUNG CANKER PRODUCED BY BACILLUS AMYLOVORUS

A, a watersprout affected with fire-blight and giving rise to the canker

Not infrequently cankers produced by the fire-blight organism (*Bacillus amylovorus*), were found at the crown of trees or on the roots near it. As is well known, the work of this organism may continue in the inner bark until it has girdled and killed the tree, or, after a period of more or less activity, these bacteria may die, leaving a sunken cankerous area,

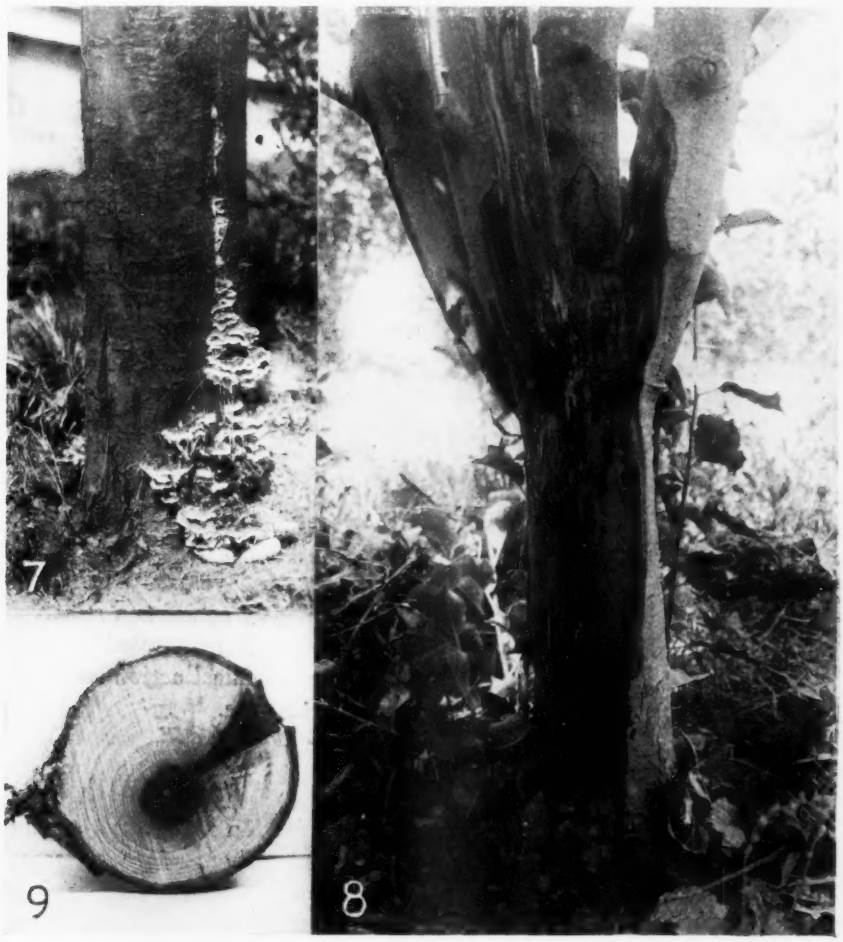

FIG. 7. POLYSTICTUS VERSICOLOR GROWING ON A PRUNE TREE

FIG. 8. FROST INJURY

The bark on the south side of the tree is peeled off and new cambium growing over practically the whole wound.

FIG. 9. A CROSS SECTION THROUGH A BRANCH KILLED BY ARSENATE OF LEAD

which develops into a distinct collar-rot condition, and, being difficult to trace to its primary cause, this is usually placed in the same category with cankers of unknown origin.

An instance of blight collar-rot is seen in figure 2, where an attempt to rescue the tree has been made by bridge grafting. The beginnings of a typical collar-rot are shown in figure 6, where a blighted water sprout (a) has been traced back to the root, forming a small canker. The bark has completely decayed over a small area, about 3.5 cm. by 10 cm., with no very definite line of demarcation between the sound and diseased tissue. Cultures on green pears from this diseased bark produced *B. amylovorus*. The wood below the bark was found to be discolored just the same as other types of collar-rot.

It is probable that the large majority of cankers found at the crown of fruit trees in Yakima Valley is due to fire-blight. This statement is difficult to prove after the organisms are dead and there is little opportunity of connecting the two except by comparison with cankers that are known to be produced by the fire-blight organism. An attempt was made to do this. Watersprouts infected with fire-blight were traced to the root and the cankers produced were carefully examined. Some of these were marked and left for several months, and re-examined. Cankers of unknown origin found on trees throughout the valley were compared with these. The result of this comparison seemed to show conclusively that many of the cankers that were previously considered of unknown origin were in reality inactive fire-blight cankers. Many of these evidently had been inactive for some time, probably several years. During this time irrigation and seepage water, which frequently contains a relatively high percentage of alkali, soaked into the wounds, penetrated into the heart-wood and discolored it, possibly by some chemical action set up by the minerals in the water or by some oxidizing enzyme.

NON-PARASITIC CONDITION

While definite organisms were found in connection with some forms of collar-rot yet more often none could be obtained, although repeated search was made for them. Most of these cases, as has just been explained, were apparently inactive fire-blight cankers. Some, however, were undoubtedly the result of other agencies. In at least two orchards the wounds at the crown of the trees were due to the work of gophers. In several others in which trees showing the characteristic foliage of collar-rot were found, an examination showed that the hardpan was only one-quarter to one-half a meter below the surface, and that the water failed to drain off readily, in some cases resulting in the roots being actually surrounded

by water for a considerable time after irrigation, often not draining off before it was time to irrigate again; while in other instances, perhaps only a few rods away, the water seeped away normally and the trees, being in such shallow soil, soon began to show drouth conditions. In cases where the water failed to drain away the young roots were killed and soon became water-soaked, turning soft in texture and brown in color. Although the effect of this trouble on the foliage is practically the same as the collar-rot, it is more properly called a "root-rot," as the trouble usually begins at the smaller roots and gradually works back, sometimes along a single root, in which case it might reach the crown before any evidence of it is shown in the leaves. In such cases it resembles the typical collar-rot and may be mistaken for it, but more frequently several roots are affected and the tree is killed before the disease reaches the crown.

Winter injury and carelessness in cultivation are common sources of injury to fruit trees at or near the crown. That frost frequently produces such injuries has been generally recognized for many years, having been reported by a number of writers (4, 5, 10, 11, 12, 25, 27, 35, 39). These wounds, if slight, may heal, but if for some reason they fail to do so, the irrigation and seepage water soaks into the wood often producing a brownish stain. The amount of discoloration is partly due to the age of the wound, which may be several years old, and partly to the amount of alkali and other minerals contained in the water that comes in contact with the wound.

Since winter injuries apparently begin at the inner phloem or cambium, the destruction of either of these causes the bark associated with it to die and begin to disintegrate. If these injuries occur at the crown they may undoubtedly give rise to a collar-rot, but winter injuries are far from being the only cause of this malady. In fact, in the Yakima Valley comparatively few of these cankers can be traced to this cause. In a certain orchard west of the city of Yakima and also one in the Naches Valley beginnings of collar-rot could be traced quite definitely to winter injury. Some of these wounds that were wrapped with pieces of cotton or old gunny sacks so as to protect the cambium from the excessive heat during the summer healed over almost completely in one season. This is shown in figure 8, in which the young cambium can be detected covering almost the whole wounded surface. These orchards, both in alfalfa, were inadvertently irrigated so late in the fall that the water had not all drained off before the frosts came. This excessive amount of water freezing in the bark near the ground caused it to split, in some cases forcing it from the wood and producing frost cankers. As the soil offered some protection to the roots these cankers did not extend more than 25 cm. below the surface. Most of the lesions, however, occurred higher up on the trunks of the trees, producing typical frost injuries.

In some cases the primary cause of the wound leading to collar-rot has been traced to defective grafting. An examination of a number of young trees from three to ten years old, killed by collar-rot, warrants the conclusion that defective unions of grafts are responsible for wounds that are the fore-runners of collar-rot.

ARSENICAL POISONING

Not infrequently there were found cankers that did not seem to be produced by any one of the conditions already mentioned. The final results were practically the same as those of winter injury or fire-blight, but the initial stages and the general effect on the bark were quite different. The resemblance of some of these cankers of unknown origin to those produced by an "ant exterminator," which was found to contain arsenic, and which when applied to the trunks of apple trees injured some and killed others, led the writer to run some experiments to determine something of the effect of arsenate of lead on growing fruit trees.[2]

In the following experiments both branches and roots were treated. The arsenate of lead was obtained from the tank of the orchardist as he was spraying. This spray consisted of an acid arsenate of lead in the form of paste with a strength of four pounds to 100 gallons of water.

Effect of arsenate of lead on branches

For convenience branches that were nearly horizontal were selected for these experiments. In some cases the bark was slightly punctured with the point of a scalpel, deep enough to reach the cambium, while in others the arsenic was applied to lenticels and in still others to the uninjured bark.

The method of treatment was practically the same in all three cases. Where a wound was made a Van Tiegham cell was fastened over it and sealed with grafting wax, care being taken not to allow any of the wax to get into the puncture. The cell, which held about 2 cc., was nearly filled with the solution of arsenate of lead and a cover glass was cemented on the top of the cell with vaseline. In four days a strip of dead bark about 2 cm. wide was found, extending from the point of puncture to the tip of the branch, which in some instances was over 8 feet long, and passing out into lateral branches, if there happened to be any in its path. The leaves along this path were killed but others on the same branch were uninjured. The branch, however, was not completely dead, for the next

[2] A review of the literature on arsenical poisoning of fruit trees is given by Swingle and Morris (34).

year the leaves came out as usual. A cross section through one of these limbs is shown in figure 9. The discolored area forms a wedge-shaped structure running into the center, the sides of which are very straight and apparently follow medullary rays. An examination of the margin of the wound shows how the new growth is attempting to heal it over.

In most cases where Van Tiegham cells were arranged over lenticels a small discolored area appeared around each. In some few instances no change could be detected, probably due to the inability of the solution to penetrate the tissue. In all cases the action seemed to be completed by the end of three or four weeks, and by the following year a decided tendency to heal over the injury was observed.

When Van Tiegham cells were placed over areas on branches where there were no lenticels nor wounds no detrimental effect on the bark was observed. These observations are practically the same as described by Swingle and Morris (37). Quite different results were obtained when the arsenic was applied to the roots.

Effect of arsenate of lead on roots

Lead arsenate obtained from the same source as that used in treating branches was applied to the roots. In these cases the soil was removed and a portion of the root as near the crown as possible was selected for treatment. The applications were made to the uninjured surface and to wounds. Both the solution and paste were applied so that the root would have the irritant action, if any, of the undissolved material as well as the dissolved. The wounds were made by cutting off small rootlets or by puncturing the bark into the cambium in a manner similar to that done in connection with the branches. Van Tiegham cells were used here at first but it was found more convenient to wrap the treated wound with several layers of cotton cloth. After the arsenate had been applied freely and the wound wrapped, the covering was well moistened with water and the soil replaced. In some instances a second application of the arsenate was made but in the majority of cases the initial treatment was the only one. Once a week for six weeks, less frequently after that, these trees were visited, the soil and bandages removed and any differences noted. In each case after the cotton covering was replaced it was well saturated with water so that the treated area was not allowed to become dry. In these examinations the chief changes observed were the increasing size of the cankers and the continued disintegration of the bark. In three weeks a definite action could be noticed where the wounds were made. The bark began to turn brownish about the wound, the discoloration increasing more rapidly longitudinally than laterally. Twelve months

after the wounds were made a typical collar-rot canker with the brown, friable bark and deeply stained wood had been developed. Three roots treated in this way are shown in figure 10. These were taken from the trees twelve months after they were first treated. An enlarged view is shown in figure 11 and cross sections of the same roots in figure 12. In specimen "C" the bark is the most markedly disintegrated and is reduced to a more or less powdery brown mass. The disease seemed to have worked faster in this case than in the others, possibly because the tree was in lower and more alkaline soil. In a few instances where the wound

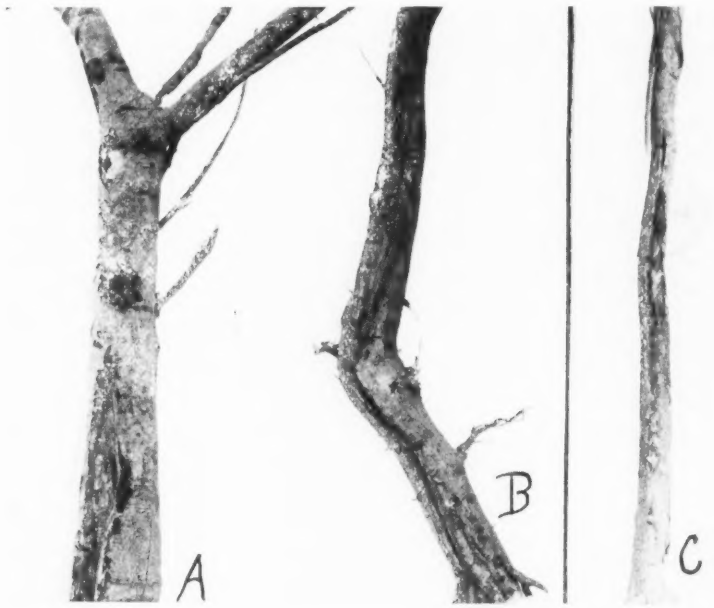

FIG. 10. THREE ROOTS SHOWING CANKERS PRODUCED BY ARSENATE OF LEAD

was allowed to dry and become aerated it gradually healed, even with the dried white arsenate paste still adhering to it, but where there was plenty of moisture with alfalfa to aid in keeping the humidity fairly constant the cankers gradually increased in size and eventually became permanent.

It is thus seen that the effect of arsenate of lead on wounded roots that are kept moist is markedly different from that on the stem, where it is almost impossible to keep treated wounds constantly in a saturated atmosphere. This is somewhat different from the conclusion arrived at by Swingle and Morris (37), who found that in Montana there was little difference in the effect on the stem and root, and "that excessive moisture

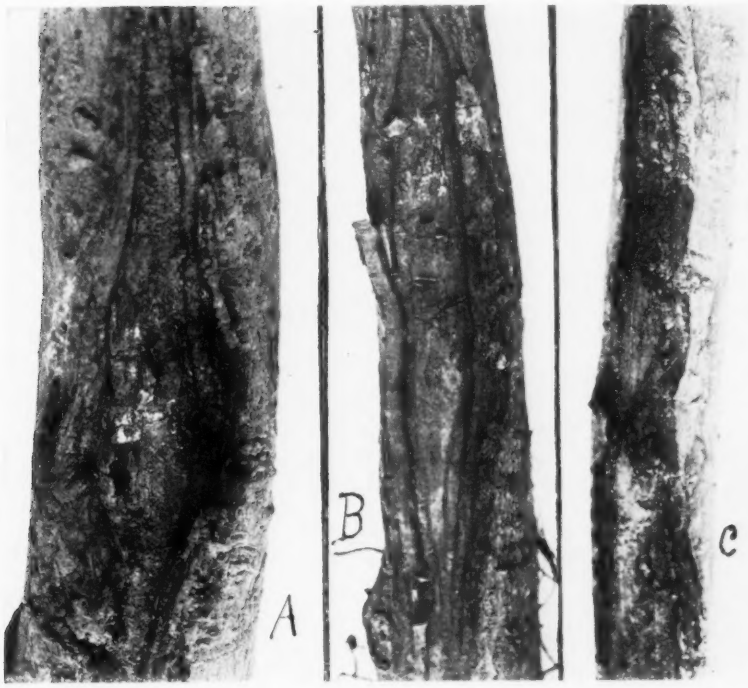

FIG. 11. AN ENLARGED VIEW OF THE CANKERS SHOWN IN FIGURE 10

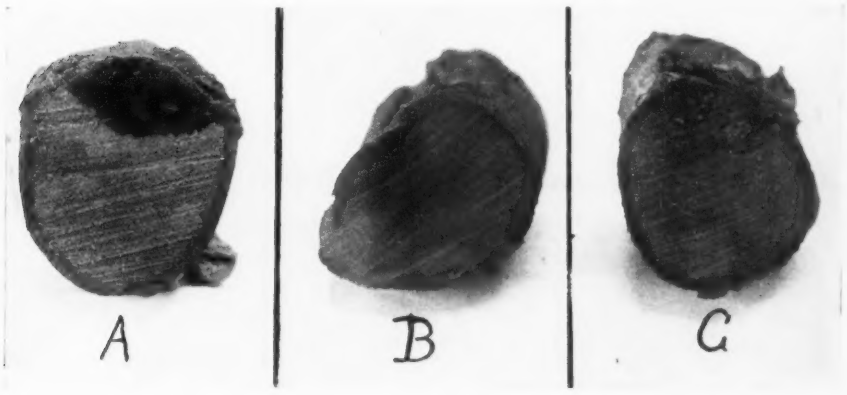

FIG. 12. CROSS SECTION THROUGH THE CANKERS SHOWN IN FIGURES 10 AND 11

and imperfect aeration did not materially influence the injury, which had usually begun in a few days and was complete within two or three weeks from the time of treatment."

When unwounded roots were treated with arsenate of lead, little or no result was observed. When applied to wounds produced by breaking off small rootlets, the injury was usually slight but definite, and in a short time, varying from two to five weeks, small brownish patches appeared, which gradually increased in size, but permanent cankers were produced less frequently than when the arsenate was applied to wounds in the bark.

WATER LENTICELS

Grossenbacher (18), working in New York State, found that trees affected with crown-rot were most prevalent in cultivated orchards and on thin, stony or gravelly soil. Quite the contrary is true in Yakima; although the disease is found in clean cultivated orchards and on deep soil, it is more prevalent on low, rich soil, especially where there is or recently has been a cover crop of alfalfa kept well irrigated.

It should be remembered that most of the experiments recorded in this article were carried on in orchards with a cover crop of alfalfa. In irrigating, the orchardist often thought of the immediate needs of the alfalfa rather than the trees. Not infrequently there were found at the crown of trees small whitish or slightly yellowish patches which were soft and spongy and which proved to be water lenticels such as are commonly seen when a willow wand is left submerged in water. When the arsenate was applied to these hypertrophied patches marked results were obtained. The large conspicuous cells collapsed, and instead of a swollen area there appeared a circular depression, the center of which eventually became a brownish powder. These affected areas in time often ran together, forming one large discolored space, which in some cases penetrated to the cambium. When these were kept moist cankers were formed. In some instances, however, after the alfalfa had been cut and the region about the treated area had become somewhat dry and aerated, the wounds, even if they had reached the cambium, dried up, leaving only small sunken areas in the bark, which did not seem to be detrimental to the tree.

It was found on further search that these excrescences were quite prevalent in orchards having a cover crop of alfalfa, which, when well irrigated, provided an almost saturated atmosphere at the base of the trees. This humidity, associated with the extreme heat of the Yakima Valley, made an excellent environment for the development of this condition, which was observed most abundantly during July and August, especially on trees that had been severely pruned.

In this search trees were not infrequently found having the bark at the crown, usually just below the ground, soft like rubber, and scattered over it a number of more or less circular, brown, dead patches in which the bark had largely disintegrated into a brownish powder. This is almost precisely what was obtained when water lenticels were treated with arsenate of lead. A photograph of a tree seven or eight years old showing this condition is shown in figure 13, where the brown powdery material has been removed to show the depth of the depression, which in the larger ones extends to the cambium.

FIG. 13. EARLY STAGES OF ARSENICAL POISONING

The lower portion of this figure at (a) shows these discolored areas run together, the outer portion of the bark becoming disintegrated, producing a brownish powder when rubbed between the thumb and finger. Beneath this is sound bark except in certain spots which have reached the cambium and discolored it slightly. On further search an almost perfect series has been observed, beginning with the isolated brown spots which enlarge, run together, and form larger brown patches but still quite shallow, while more advanced stages penetrate entirely through the bark, discoloring the wood just beneath.

These observations show that while the early stages of the lesions caused by frost and winter injury, as shown by Grossenbacher (22), begin at the cambium or inner phloem and eventually cause the bark, which subsequently dies, to be separated from the wood, the lesions caused by arsenate of lead have their initial stages at the outer bark, either at some wound or at such hypertrophies as water lenticels. The corrosion thus begun at the surface of these excrescences gradually destroys the cells and eventually reaches and kills the cambium, producing a permanent canker.

The results of the present investigation as well as those recorded by Headden (23, 24) and Swingle and Morris (37), show conclusively that arsenate of lead will produce definite injuries to the bark of fruit trees. There is little doubt also that a typical collar-rot condition can be produced by the application of commercial arsenate of lead to fresh wounds or water lenticels found at or near the crown. Because cankers are produced artificially when a considerable amount of arsenate is used is not, alone, an absolute proof that it does so in nature, when there is a much smaller amount present. Yet, when a series of conditions practically identical with the artificial injuries are found in nature, it seems highly probable that the arsenate of lead such as is commonly used by orchardists is capable of becoming the primary cause of these cankers.

The writer has produced similar cankers on fruit trees by using copper sulphate, creosote, turpentine, etc. Even with such weak toxic substances as boric acid, potassium chloride, borax, and even sodium chloride, cankers can be made by using concentrated solutions on fresh wounds. This would indicate that the presence of alkali in the soil may perhaps greatly augment the work of the arsenate, not only in increasing the solubility of the arsenate, as shown by Headden (23), but even by its chemical effect on the living tissues of the tree. Considerable alkali, which in the Yakima Valley always contains bicarbonate and carbonate of sodium and frequently the sulphate and the chloride, is always present in this soil. A concentrated solution of these compounds was made and applied to fresh wounds of apple trees in Seattle, producing definite and serious injuries to the trees. It would thus appear that although arsenate of lead is capable of producing permanent cankers itself, yet there is often a combination of chemical factors at work producing this result, and of these the most important one is probably the arsenic.

Moreover, observational evidence leads the writer to the belief that moist soil, especially if alkali is present, when kept more or less constantly in contact with a wound, contributes more toward producing a permanent canker than has been generally accepted. The fact that a wound, from whatever cause, fails to heal indicates that the cambium over the surface is dead. Water containing a relatively high percentage of alkali and

probably other minerals from the soil may then gradually soak through the dead tissue into the heart of the tree, following along the dead medullary rays, discoloring the cell walls and eventually producing a collar-rot canker.

WOOLY APHIS

The development of the primary lesions into typical collar-rot conditions depends largely on the persistence of the factors which prevent the healing of the wounds. It would appear from the present observations that one of the chief agents at work hindering these wounds from healing is the woolly aphis. These insects were found in connection with nearly all forms of collar-rot, but especially in the earlier stages of its develop-

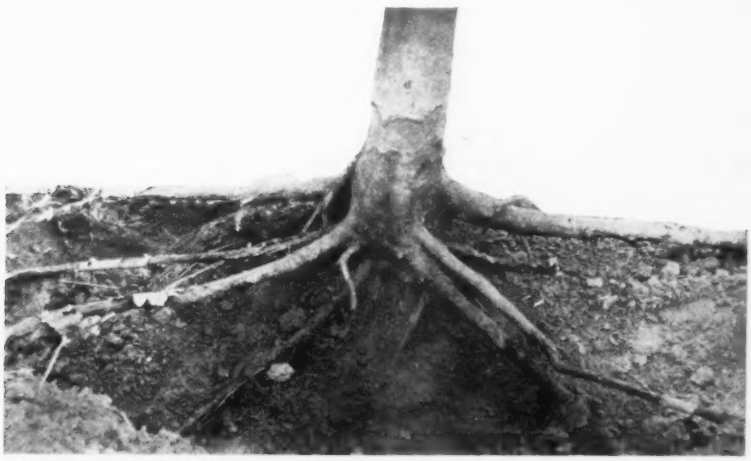

FIG. 14. A TREE COMPLETELY GIRDLED BY COLLAR-ROT

ment. They readily attack apple trees of all ages and varieties but in general the young trees suffer most. As is well known, the favorite location for their operations is at the crown of the tree just where collar-rot develops. In 75 per cent of the young stages of collar-rot examined by the writer, woolly aphids were found in large numbers. In these cases it would appear that the injury having been made, the aphids occupy the wound, persistently attacking the fresh margin and thus apparently becoming an important factor in preventing it from healing. To what extent these aphids are capable of producing the original wound was not determined, but it is quite evident that when once an abrasion is made their activities tend to prevent the wound from healing, thus contributing towards a permanent canker.

TREATMENT

In attempting to treat collar-rot the soil was removed, laying bare all the affected parts. These were cleared of all dead bark and thoroughly washed with water in order to obtain a clean surface and then disinfected. A weak solution of lysol, about 1 per cent, was found the most effective, although corrosive sublimate, weak copper sulphate, Bordeaux mixture, lime-sulphur, and formaldehyde were also used. The superiority of the lysol seems to be due to the fact that it penetrates the tissue of the tree a certain distance without any apparent injury. Its effect is thus more permanent. After the wounds were all treated they were left exposed to the air for two or three months. Before the rains came in the autumn the excavations were filled with sand, gravel or cinders, thus facilitating drainage.

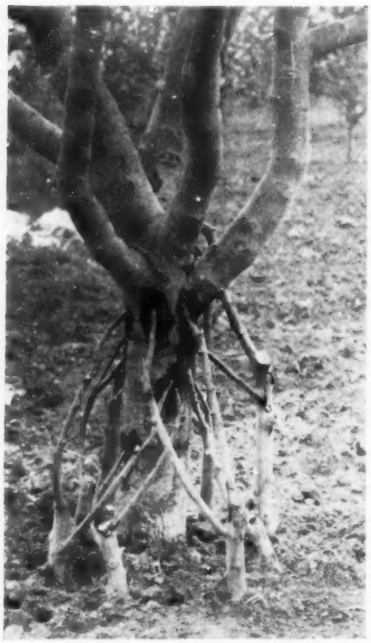

FIG. 15. AN ATTEMPT TO SAVE THE TREE SHOWN IN FIGURE 14, BY PLANTING SEEDLINGS AROUND IT AND GRAFTING THE ENDS OF THE BRANCHES INTO THE SOUND BARK ABOVE

It would be wise for an orchardist to go over his whole orchard once in two or three years with a trowel or shovel, remove the soil from the crowns, and determine if the collar-rot had started. If there is any indication of the disease, the dead bark should be removed and the wound disinfected and finally left exposed to the air for several months.

Bridge grafting has been indulged in at times in an attempt to save valuable trees. The number of scions used varied with the size of the tree, frequently too few being used. The unions were made just below the bark in the sound tissue of the stem above and of the root below the canker. Occasionally when too much of the root was destroyed, young seedlings were planted around the tree, as shown in figure 15, and the ends of the branches cut off and grafted into the sound cambium above. The same tree just before the operation is shown in figure 14. It can be seen that the greater part of the root system is dead, hence the necessity for using young seedlings. Bridge grafting as a method of treatment for collar-rot can hardly be said to be a success. Although a fair percentage

of the trees live, they are seldom thrifty, and both the quality and quantity of the fruit they produce are inferior. Moreover, since a large space of the dead stem of the tree is usually left exposed, the tree eventually contracts heart-rot, which forecasts its early death. In the majority of cases, therefore, it would seem more profitable in the long run, where the injury is severe, to remove the affected tree and after a year or two to plant another.

SUMMARY

1. It would appear that the only condition essential for the development of the so-called collar-rot or crown-rot is a permanent wound of the bark at or near the crown of a fruit tree. As far as the results are concerned it matters little as to the cause of these lesions.

2. A number of different factors may be involved, either as primary causes or contributing causes, in the development of the collar-rot condition.

3. Most of the numerous attempts to isolate definite organisms from the collar cankers have resulted negatively.

4. In a few cases such organisms as *Armillaria mellea*, *Polystictus versicolor*, and *Bacillus amylovorus* have been definitely isolated.

5. The great majority of cankers whose primary cause could not be directly determined were probably initiated by the fire-blight organism, a conclusion arrived at by a comparison of such cankers with those known to have been begun by *B. amylovorus*.

6. Other causes, such as gopher injury, cultivation injury, frost or winter injury, defective graft unions, hard-pan root injury, and arsenate of lead injury, may be responsible for the initial development of collar lesions.[3]

7. The development of the primary or original lesion into a typical collar-rot condition depends upon the persistence of factors which prevent the healing of the wound.

[3] Since this article was written further work on this disease has brought out evidence that the gradual corrosion of the bark under certain conditions might be the result of a more or less continuous oxidation, the oxidases acting either as the primary causes or contributing factors. This hypothesis is based on the following observations:

1. There is an oxidase present in the bark as shown by the bluish color produced by guaiacum.

2. The diseased bark is less acid than the healthy bark.

3. Oxidases tend to work more rapidly where the acidity is weak. The presence of constant moisture more or less charged with alkali reduces the acidity in the immediate vicinity of the abrasion, thus making the conditions more favorable for oxidation.

8. Observational evidence leads the writer to the belief that there are at least two important factors at work in preventing the normal healing of the crown-lesions and therefore in promoting the characteristic disintegration of the tissue known as collar-rot. These are the presence of the woolly aphids and a saturated condition of the soil in constant contact with the primary lesions, particularly if the soil water is charged with alkali salts.

9. The only method of treatment thus far devised is the cutting out of diseased tissue, the washing of the wound, disinfecting it, and finally leaving it exposed to the air. Where injury is severe when discovered, removal of the tree is probably wise.

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LITERATURE CITED

(1) BAILEY, I. W.: Oxidizing enzymes and their relation to sap stains in lumber. Bot. Gaz. **50**: 142–147. 1910.

(2) BALL, E. D.: Is arsenical spraying killing our fruit trees? Jour. Econ. Ent. **2**: 142–148. 1909.

(3) BALL, E. D., TITUS, E. G., AND GREAVES, J. E.: The season's work on arsenical poisoning of fruit trees. Jour. Econ. Ent. **3**: 187–197. 1910.

(4) BURRILL, T. J.: Climatal destruction of orchard trees. *In* 13th Rpt. Univ. Illinois **1884**/**86**: 283–293. 1887.

(5) CLINTON, G. P.: Diseases of cultivated plants in Connecticut. Winter injury. Connecticut Agr. Expt. Sta. 27th Ann. Rpt. [**1902**] **03**: 303–304. 1904.

(6) CLINTON, G. P.: Notes on fungous diseases, etc., for 1904. Apple-winter injury. Connecticut Agr. Expt. Sta. 28th Ann. Rpt. [**1903**] **04**: 312–313. 1905.

(7) CLINTON, G. P.: Notes on fungous diseases for 1906. Winter injury and canker, Sphaeropsis malorum Pk. Connecticut Agr. Exp. Sta. 30th Ann. Rpt. [**1905**]/**06**: 310–311. 1907.

(8) CLINTON, G. P.: Notes on fungous diseases for 1908. Chestnut bark disease, Diaporthe parasitica Murr. Connecticut Agr. Exp. Sta. 31st/32d Ann. Rpts. [1906] 08: 879–890. 1908.

(9) CRABILL, C. H.: Note on apple root-rot in Virginia. Phytopathology **6**: 159. 161, 1 fig. 1916.

(10) CRAIG, JOHN: Observations and suggestions on the root-killing of fruit trees, Iowa Agr. Expt. Sta. Bul. 44: 179–213, illus. 1900.

(11) EMERSON, R. A.: Experiments in orchard culture. Nebraska Agr. Expt. Sta. Bul. 79; 33 p., 12 fig. 1903.

(12) EMERSON, R. A.: The relation of early maturity to hardiness in trees. Nebraska Agr. Expt. Sta. Ann. Rpt. **19**: 101–110, illus.

(13) FROMME, F. D., AND THOMAS, H. E.: Black rootrot of the apple. Jour. Agr. Research **10**: 163–174, 1 fig., pl. 15–17. 1917.
Literature cited, p. 172–173.

(14) FROMME, F. D., AND THOMAS, H. E.: The root-rot disease of the apple in Virginia. Science n. s. **45**: 93. 1917.

(15) FROMME, F. D., AND THOMAS, H. E.: A Xylaria root-rot of the apple. (Abstract.) Phytopathology **7**: 77. 1917.

(16) GIDDINGS, N. J.: The collar blight of apples. Rpt. West Virginia Dept. Agr. **29** 15-19, 1 fig. 1913.

(17) GILLETTE, C. P.: New sprays for the codling moth. Jour. Econ. Ent. **3**: 29, 1910.

(18) GROSSENBACHER, J. G.: Crown-rot, arsenical poisoning and winter-injury. New York State Agr. Expt. Sta. Tech. Bul. 12: 369–411, pl. 1–8.

(19) GROSSENBACHER, J. G.: Crown-rot of fruit trees. Field studies. New York State Agr. Expt. Sta. Tech. Bul. 23: 36. 1912.

(20) GROSSENBACHER, J. G.: Some neglected phases of phytopathology. Phytopathology **5**: 155–162. 1915.

(21) GROSSENBACHER, J. G.: Some bark diseases of citrus trees in Florida. Phytopathology **6**: 29–50, illus. 1916.
Bibliographical footnotes.

(22) GROSSENBACHER, J. G.: Crown-rot of fruit trees. Histological studies. Amer. Jour. Bot. **4**: 477–512, pl. 21–27.
Bibliographical footnotes.

(23) HEADDEN, W. P.: Arsenical poisoning of fruit trees. Colorado Agr. Expt. Sta. Bul. 131, 27 p., 7 pl. 1908.

(24) HEADDEN, W. P.: Arsenical poisoning of fruit trees. Colorado Agr. Expt. Sta. Bul. 157, 56 p., illus. 1910.

(25) NELSON, AVEN: The winter-killing of trees and shrubs. . . . . Wyoming Agr. Expt. Sta. Bul. 15: 213–222. [1893.]

(26) ORTON, C. R., AND ADAMS, J. F.: Collar-blight and related forms of fire-blight. Pennsylvania Agr. Expt. Sta. Bul. 136, 23 p., 13 fig. 1915.

(27) PADDOCK, WENDELL: The New York apple-tree canker. New York State Agr. Expt. Sta. Bul. 185: 205–213, 4 pl. 1900.

(28) PADDOCK, WENDELL: Plant disease of 1901. Colorado Agr. Expt. Sta. Bul. 69, 23 p., 9 pl. 1902.

(29) PICKETT, B. S.: The blight of apples, pears, and quinces. Illinois Agr. Expt. Sta. Circ. 172, 10 p., illus. 1914.

(30) REED, H. S., AND CRABILL, C. H.: Notes on plant diseases in Virginia observed in 1913 and 1914. Virginia Agr. Expt. Sta. Tech. Bul. 2, 58 p., 17 fig. 1915.
Also in Virginia Ag. Expt. Sta. Ann. Rpt. **1913/14**: 37–58. 1915.

(31) SCHRENK, HERMANN VON: A root rot of apple trees caused by Thelephora galactina, Fr. Bot. Gaz. **34**: 65. 1902.

(32) SELBY, A. D.: Some diseases of orchard and garden fruits. Ohio Agr. Expt. Sta. Bul. 79: 94–141, illus., pl. 9. 1897.

(33) SELBY, A. D.: A brief hand-book of the diseases of cultivated plants in Ohio. Ohio Agr. Expt. Sta. Bul. 214: 307–456, illus. 1910.

(34) SMITH, R. E., AND SMITH, ELIZABETH H.: California plant diseases. California Agr. Expt. Sta. Bul. 218: 1039–1193, illus. 1911.

(35) STEWART, F. C., ROLFS, F. M., AND HALL, F. H.: A fruit disease survey of western New York in 1900. New York State Agr. Expt. Sta. Bul. 191: 291–331, 6 pl. 1900.

(36) SWINGLE, D. B., AND MORRIS, H. E.: A preliminary report on the effects of arsenical compounds upon apple trees. Phytopathology **1**: 79–93, pl. 16–17. 1911.

(37) SWINGLE, D. B., AND MORRIS, H. E.: Arsenical injury through the bark of fruit trees. Jour. Agr. Research **8**: 283–318, pl. 79–84. 1917.

(38) WHETZEL, H. H.: The blight canker of apple trees. New York Cornell Agr. Expt. Sta. Bul. 236: 99–138, illus.

(39) WHIPPLE, O. B.: Report of the field horticulturist for 1906. Colorado Agr. Expt. Sta. Bul. 118, 16 p. 1907.

(40) WILCOX, E. M.: A rhizomorphic root-rot of fruit trees. Oklahoma Agr. Expt. Sta. Bul. 49: 32 p. 11 pl. 1901.

(41) WOLF, F. A., AND CROMWELL, R. O.: Xylaria rootrot of apple. Jour. Agr. Research **9**: 269–276, 3 fig., pl. 3. 1917.

A PRELIMINARY STUDY OF THE PURPLE LEAF SHEATH SPOT OF CORN

L. W. Durrell

With Six Figures in the Text

The purple sheath spot of corn is a conspicuous and universally present spot disease, yet its cause has never been definitely determined. It is the purpose of this paper to present observations and experiments demonstrating the cause of this disease.[1]

The prevalence and distribution of the disease is suggested by Burrill's (2) statement, which reads as follows: "From observations now made it appears that the disease is a very prevalent one and probably has existed during the time that corn has been grown on the continent." It has been observed in Iowa by the author and members of the station staff over a period of four years. During the latter part of the summer of 1918, counts on 9620 stalks in representative fields showed 86 per cent badly spotted with the purple leaf sheath spot.

The characteristic symptom is a purple or brown spotting or blotching of the leaf sheaths and husks (fig. 1). This condition develops rapidly on all plants after flowering. Examination of the blotches reveals a darkened, disintegrated condition of the tissues on the inside of the sheath. In the center of the larger blotches one usually finds old pollen sacs that have been dropped from the flowers above. In the center of the smaller spots one finds pollen grains that have been washed or shaken down inside of the sheath, as shown in figure 2.

The purple sheath spot of corn was first definitely described in 1889 by Burrill (2 and 3), who attributed it to a bacterial organism. He writes of it as follows: "The first indication of the disease in a field . . . . is the dwarfed condition of the young plants. . . . . It is found in the rich spots rather than in those of poorer quality. In many cases it is upon the lowest ground. . . . . Upon closer observation it is found that young diseased plants, besides being smaller . . . . are uniformly yellowish in color. . . . . The bottom portion of the stalk is likewise affected. . . . . If split longitudinally . . . .

[1] The author wishes to acknowledge the many helpful suggestions made during the progress of this study and the preparations of the manuscript by Dr. I. E. Melhus.

the inner tissue of this lower part is seen to have a uniform dark color. . . . . After midsummer, especially, the disease becomes apparent through discolorations of . . . . the sheaths, which closely invest the stalk. These leaf sheaths become variously spotted as observed from the exterior. Sometimes the watery-brown portions are mere specks; sometimes large irregular patches. . . . . Occasionally there is a little reddish color. . . . . If now these affected leaves are stripped from the stalk, it will be found that evidence of the injury is more conspicuous on their innerside. The injured patches . . . . have a more watery appearance and sometimes are more or less smeared with some gelatinous substances. . . . . ."

In 1888 Kellerman and Swingle (7) discovered a bacterial spot of sorghum, and sent specimens to Burrill for study. This apparently led Bur-

FIG. 1. PURPLE LEAF SHEATH SPOT OF CORN

rill to the investigation of similar spots on corn sheaths, with the subsequent publication of the above description. At this time, too, interest was aroused over a disease of cattle, apparently caused by eating diseased corn stalks. The sheath spot of corn was one of the diseases thought to be responsible; experiments, however, did not confirm this assumption. Billings (1) of Nebraska, in correspondence with Burrill on this subject, writes as follows: "The malady as affecting this staple crop has not been fully worked out because for some unknown reason the attempts to communicate the disease to healthy plants by application of culture materials containing the living organism obtained from diseased plants have not been successful." He further states that no results were obtained by inoculating plants with an organism that he had isolated and considered specific for the cattle disease. Not until August, 1889, did Burrill obtain

satisfactory results from inoculations with the organism he had isolated from the corn spots, and his success even then was slight, except where punctures were made in the surface of the leaf sheaths.

In the light of our present knowledge of corn diseases, the description he furnishes throws some doubt upon the work, for it would appear that the symptoms as described have been associated also with other corn diseases, such as corn wilt. Writing of corn wilt Smith (10) says: "The

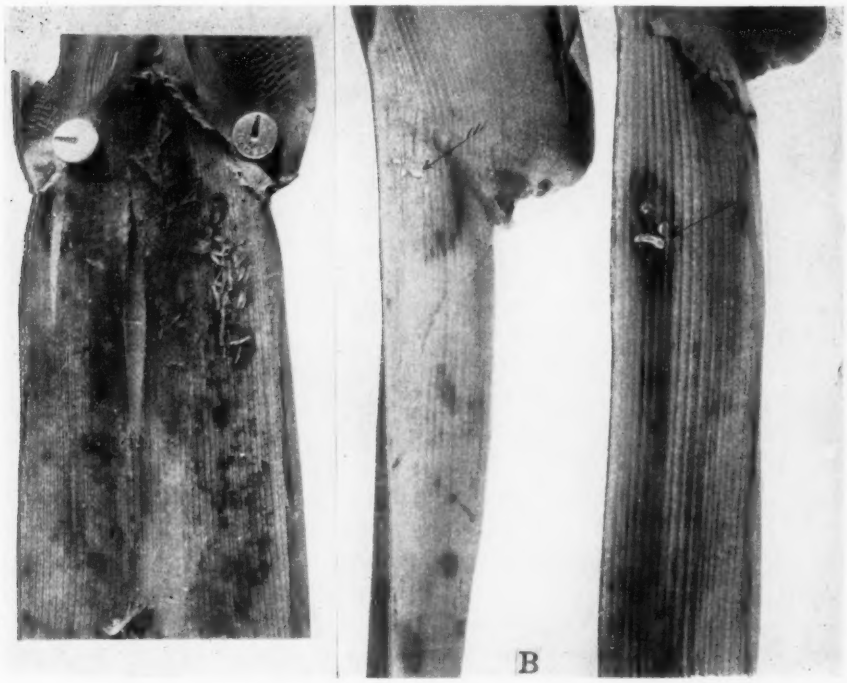

FIG. 2. (*A*) Inner surface of corn sheath, showing adhering pollen sacs that have dropped past loosened ligule, and the resulting discolored areas of infection. (*B*) Pollen sacs on inside of corn sheath, and the surrounding fungus invaded areas.

history of this disease begins with Stewart's bulletin (1897), unless we are to assume that Burrill's bulletin on 'A Bacterial Disease of Corn' (1889) relates in part to the same subject. . . . . Some parts of his description correspond very well to Stewart's disease, e.g., irregular distribution of the disease in the field; dwarfing; brown nodes; the base of the stalk most badly affected; death of the leaves as a whole . . . . . ; but if he really had this disease under observation, why is there no mention of the most conspicuous sign, i.e., yellow slime in the internodal

bundles? . . . . Along with these signs, as a part of the same disease, are mentioned other less characteristic signs, i.e., decaying roots . . . . .; brown spots on the leaf sheaths, most conspicuous within, and occasionally bordered by red. These brown spots are 'half rotten' discolorations, the disease in this stage being evenly distributed through this field. . . . . The description of the organism which he isolated and with which he obtained infections is what finally decided me to exclude it. This was a white peritrichiate schizomycete subsequently identified by Dr. Theobald Smith as *Bacillus cloacae*."

The dwarfing and decaying of roots of the corn plants described by Burrill suggests the possibility that he had under observation the disease discussed by Hoffer and Holbert (6) in their study of root and ear rots.

Further mention of the sheath spot is made by Rosen (8) in connection with his studies of a bacterial rot of corn. He writes: "The only other diseased condition noted was a dark brown spotting of husks, leaf blades and sheaths. It is possible that this represents a type of secondary injury due to the pathogen responsible for the root rot. It may be mentioned that aphids have been found associated with some of these spots, which may act as carriers of the disease producing organism, also that small, purplish-brown spots resembling the spots found on plants growing in the field have been produced by artificial inoculation with the organism causing root rot."

It is obvious from the above brief review that the cause of the purple leaf sheath spot was not definitely demonstrated by Burrill and has not received serious consideration by subsequent workers.

The author (4) presented an abstract at the St. Louis meeting of the American Phytopathological Society, briefly describing the cause of this disease on the leaf sheaths. In this paper it is planned to present a more complete discussion as to the nature of the purple leaf spot of corn.

TIME AND APPEARANCE OF SHEATH SPOT AS RELATED TO DISTRIBUTION OF POLLEN

The disease appears late in the season at the time of, or shortly after, blossoming of corn. In the plots of known ancestry in the experiment station fields, where counts were made, practically all of the stalks exhibited spotted sheaths after flowering, and the development of the disease advanced rapidly in all parts of the field after the dropping of the pollen.

This observed fact suggested a possible association of pollen with the disease. Examination of the tissues of the sheath next to the stalk, as previously mentioned, showed a rotted condition, and in every case

either pollen sacs or pollen grains were found in the center of the spots (fig. 3).

The leaf sheath of corn completely encloses the stalk with over-lapping edges, and when young the ligule fits tightly. As the plant matures, however, this grasp is relaxed, and the action of the wind on the leaves loosens the ligule and affords a means of entrance for spores, dust, pollen, and organisms of various kinds. Pollen and pollen sacs, falling from the tassel above, light on the leaves in great masses; they are readily washed past the loosened ligule and lodge within the sheath. Centrifuged washings from many leaves reveal numerous spores of different fungi, which, together with the pollen, lodge within the moist enclosure of the leaf sheath. Earlier in the season, the rapid growing stalk forces out anything introduced within the sheath. Bits of fungi on media introduced within the sheath in the evening have been found above the ligule the next day. After flowering, however, there is practically no elongation of the stalk; consequently, pollen and spores remain within the moist enclosure of the sheath, where excellent conditions are furnished for organisms to begin growth on the food stored in the pollen. When this is exhausted they attack the tissues of the sheath. The effect on the host tissue is a corrosion and discoloration of the tissues of the sheath surrounding the stalk. The stalk and the outer part of the leaf sheath are scarcely ever attacked. This is readily explained by the presence of stomata on the inner surface of the sheath, affording an entrance for the organisms, together with the difference in thickness of the cell walls of relative parts, as shown in figure 4.

FIG. 3. ENLARGED VIEW OF POLLEN SACS AND RADIATING AREAS OF INFECTION

Produced in greenhouse

INFECTION EXPERIMENTS

Acting on the observational evidence that introduced pollen grains or pollen sacs aid infection, experiments were conducted in the field to exclude these. Cotton gauze was wrapped around the stems of a number

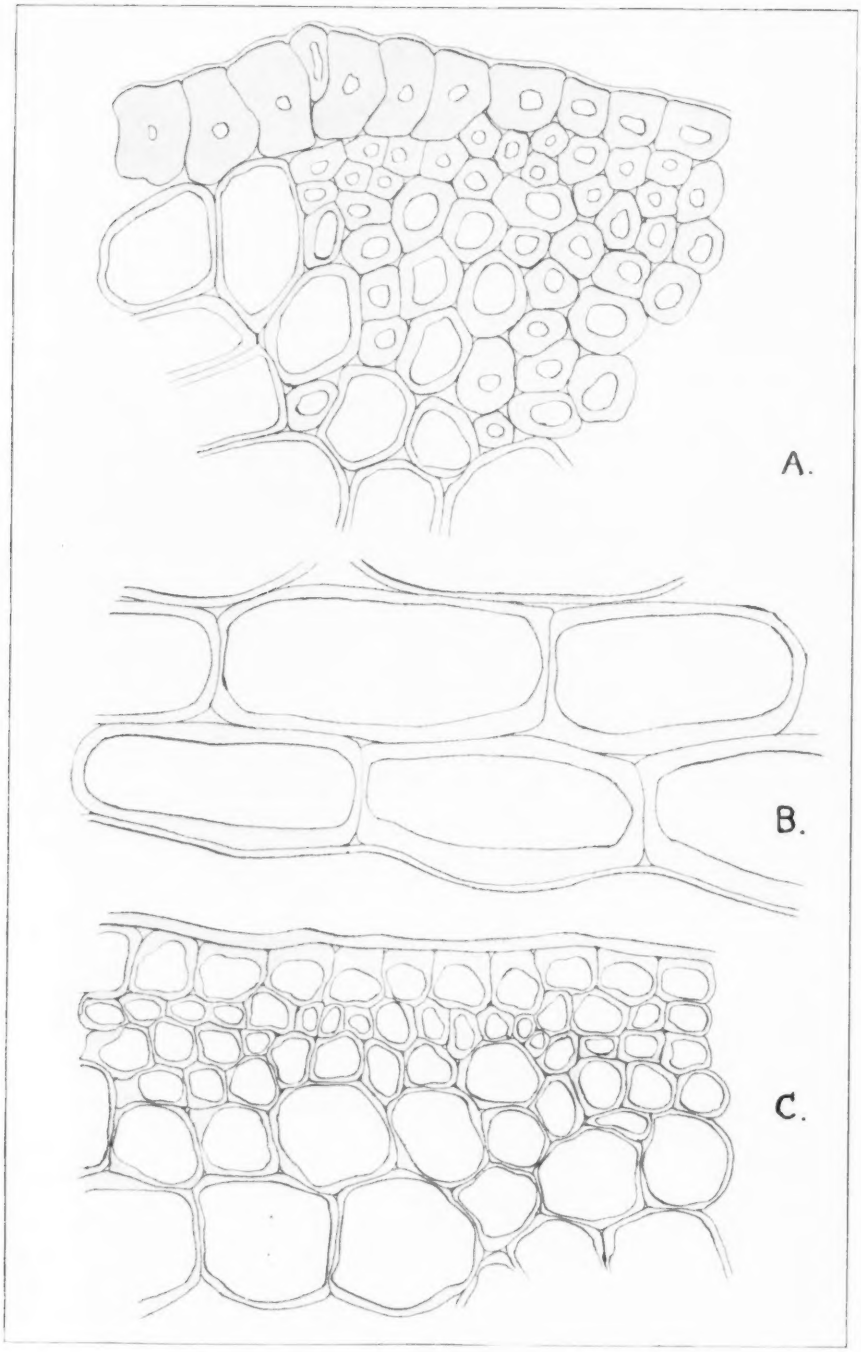

FIG. 4. CAMERA LUCIDA DRAWINGS OF SECTIONS OF SHEATH AND STALK OF CORN (*A*) Hard outer surface of sheath, (*B*) thinner walled inner surface of sheath, (*C*) hard surface of stalk adjacent to sheath.

of plants just above the ligule and tied tightly so as to exclude any pollen falling from above. Where it was tied tightly enough, no spot resulted on the sheaths. Other stalks were detasseled and covered with tall cloth houses to exclude pollen. Under this condition no spotting of the sheaths resulted, while all the neighboring stalks were infected.

In the green house results were comparable to those noted in the field Here experiments were made excluding pollen from the leaf sheaths by. growing the plants away from any source of pollen. Plants thus grown developed no spots on the sheaths. Further, pollen and pollen sacs, direct from the tassel, were introduced within sheaths of corn grown away from any source of pollen and free from sheath spot. Following such in-

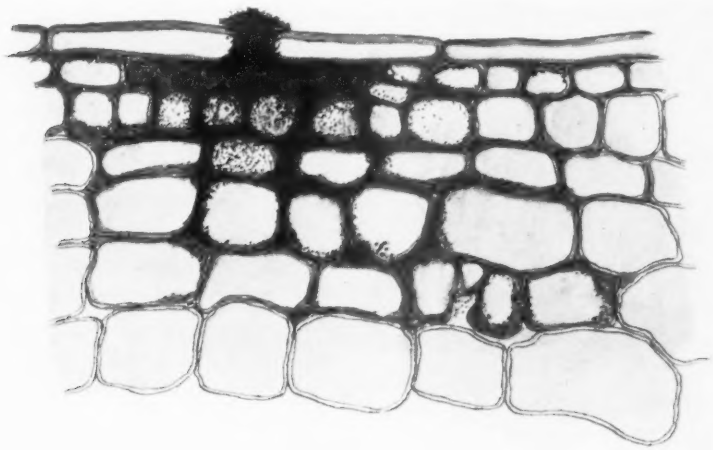

FIG. 5. SECTION OF SMALL DISEASED SPOT ON SHEATH OF SWEET CORN

Showing invasion of bacteria from excretions of aphids and the resulting corrosion of the tissues.

troductions the sheath spot soon developed, species of Penicillium usually being the causal agent.

Different organisms were obtained from plantings made from the leaf spot as it developed in the field. From 304 plantings of diseased tissue, species of Penicillium and Fusarium, and several bacteria were obtained. The Fusarium was the most prevalent. This suggested that perhaps more than one organism attacked the sheaths. Based on this assumption, pollen sacs were dusted with spores of the above mentioned fungi, which had been isolated in April, 1918, and placed inside the leaf sheaths of corn growing in the greenhouse. It was found possible to produce the sheath spot with all of the above named organisms. However, it was felt that

these experiments were not sufficiently convincing and accurate, due to the unknown agents used as inoculum. In April and May of 1919, corn growing in the greenhouse was inoculated with bacteria and fungi of known species.

Two species of bacteria were used, *Bacillus subtilis* and *B. coli*, and four species of fungi, *Fusarium culmorum*, *Rhizopus nigricans*, *Penicillium glaucum*, and *Helminthosporium sativum*. All of these organisms in many trials produced necrotic spots on the inner surface of the leaf sheaths if they were applied growing on small bits of media or if their spores were dusted on pollen sacs. The necrotic spots on the inner surface of the sheaths caused the purple sheath spot so prevalent in the field.

These infection experiments of two seasons seem to indicate that many organisms will corrode and discolor the leaf sheaths of corn if given desirable media, such as pollen, to start growth on. Sections of the spots produced artificially by these various fungi show plainly the invasion of the organisms (figs. 5 and 6). When spores or masses of mycelium were placed under the leaf sheaths without pollen or pollen sacs, no spots resulted. If, however small bits of agar were used instead of pollen, purple spots often resulted. Since the organisms causing the spots are mainly saprophytes, it is apparently necessary for them to be given a start on stored food, such as pollen or agar. The organisms seem to kill the cells of the corn plant sufficiently to allow subsequent invasion.

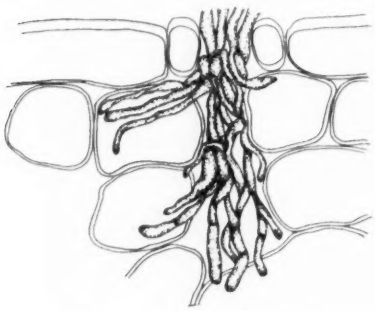

FIG. 6. MYCELIUM OF MACROSPORIUM SP. ENTERING STOMA OF CORN SHEATH

It has been noted further that the sugary excretions of aphids act in the same capacity as pollen; organisms grow on it and later penetrate the corn tissues. Many instances of distribution of organisms by insects are extant, and the carrying of spores by ants is well known. Forbes (5) thought the chinch bug in some way responsible for the corn sheath spot. In the case under consideration there seems to be little doubt that ants aid in the production of the sheath spot. It was frequently observed that the ants placed the aphids within the corn sheath even when it seemed quite tight. In this way they act as carriers of bacteria or spores. The punctures of the aphids afford added means of entrance for the organisms.

SUMMARY

Corn is universally affected by a disease producing purple blotches and spots on the leaf sheaths.

These spots are an external manifestation of necrosis of the tissues beginning on the inside of the sheaths, appearing only after the pollen has fallen. On the dead spots are pollen and pollen sacs, which seem to serve as media for various bacteria and fungi. These organisms seem to live saprophytically on the pollen at first, and later attack the adjacent host cells of the corn.

Sections of purple spots show stomatal invasion of bacteria and fungi and subsequent necrosis of the tissues.

The introduction of pollen or pollen sacs dusted with spores of molds and bacteria within the leaf sheaths produces the spots.

Spores of fungi alone when introduced within the sheaths do not produce spots, but if bits of agar are introduced spotting occurs.

Exclusion of pollen from the leaf sheaths prevents the development of the purple leaf sheath spot.

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LITERATURE CITED

(1) BILLINGS, F. S.: Original investigations of cattle diseases of Nebraska. II. The "corn-stalk" disease on cattle. Nebraska Agr. Expt. Sta. Bul. 7/10; 165. 1889.

(2) BURRILL, T. J. A bacterial disease of corn. Illinois Agr. Expt. Sta. Bul. 6: 165–175. 1889.

(3) BURRILL, T. J. A bacterial disease of Indian corn. Proc. 10th Ann. Meeting Soc. Prom. Agr. Sci. 1889: 19–27. 1889.

(4) DURRELL, L. W.: The purple sheath spot of corn. (Abstract.) Phytopathology **10:** 54. 1920.

(5) FORBES, S. A.: Bacterium a parasite on chinch bug. Amer. Nat. **16:** 824–825. 1882.

(6) HOFFER, G. N., AND HOLBERT, J. R.: Selection of disease-free seed corn. Indiana Agr. Expt. Sta, Bul. 224. 16 p., illus. 1918.

(7) KELLERMAN, W. A., AND SWINGLE, W. T.: Sorghum blight. Kansas Agr. Exp. Sta. 1st Ann. Rpt. 1888: 281–302. 1889.

(8) ROSEN, H. R.: A bacterial root-rot of field corn. Arkansas Agr. Exp. Sta. Bul. 162, 7 p., 4 pl. 1919.
Bibliography, p. 6.

(9) SMITH, ERWIN F.: Completed proof that P. stewartii is the cause of the sweet corn disease of Long Island. Science n. s. 17: 457. 1903.

(10) SMITH, ERWIN F.: Bacteria in Relation to Plant Diseases. Vol. 3. Washington, D. C., 1914. (Carnegie Inst. Washington Pub. 27, vol. 3.)

PHYTOPATHOLOGICAL NOTES

The summer field meeting of the American Phytopathological Society.—The American Phytopathological Society has successfully continued the custom inaugurated last year of holding a summer field meeting under the management of the Advisory Board of American Plant Pathologists. Last year's meeting was held at Riverhead, Long Island, June 24–27, 1919, with special reference to potato diseases. The meeting this year, which was especially concerned with apple and peach diseases, was held August 3–6, beginning at Staunton, Va., and extending northward through West Virginia and Maryland to Gettysburg, Pa., one day being spent in each of the four states.

Local arrangements were in charge of the plant pathologists of the states visited, in coöperation with the county agents, the local associations of fruit growers and other local organizations. These local committees were markedly successful in anticipating all needs and in providing all necessary facilities for the transportation and entertainment of the large party of visitors. Many demonstrations and exhibits of fruit diseases were prepared as well as special comparative spraying and dusting experiments on the control of various diseases. Transportation from point to point was by automobiles provided by the growers' associations, with occasional short journeys by rail.

August 3 was devoted to a tour of the Staunton-Harrisonburg district in Virginia, and included the inspection of interesting demonstrations of apple root rot and cedar rust, and comparative dusting and spraying experiments for control of various apple diseases. The party spent August 4 in Berkeley County, West Virginia, noting the effects of cedar eradication, visiting orchards where dusting and spraying experiments were in progress, and inspecting demonstrations of collar blight and other diseases. Visits were also made to the experimental packing plant at Inwood, and to the West Virginia pathological weather instrument station near Martinsburg. August 5 was spent in the vicinity of Hagerstown, Md., and was largely devoted to peach diseases and their control by dusting and spraying, some attention also being given to truck crop diseases. On August 6 the party visited the Field Laboratory of the Pennsylvania Agricultural Experiment Station at Arendtsville, Pa., and inspected experiments in progress in that region under direction of the laboratory staff on the control of apple diseases and insects. The conference adjourned at Gettysburg but on

August 7 a portion of the party continued by automobile to Philadelphia visiting the rich agricultural districts of Lancaster County and inspecting the tobacco experiments in progress there.

About seventy-five plant pathologists, including a number of horticulturists, entomologists and other specialists representing the United States Department of Agriculture, and the experiment stations of the United States and Canada attended the conference. In addition, the Federal Government detailed specialists from the Geological Survey, the Bureau of Soils and the Bureau of Chemistry to attend the conference and participate in the discussions on the relation of soils to plant diseases. The evenings were devoted to informal meetings, where the growers joined the visiting scientists in reviewing the day's tour and in discussing fruit disease problems. An unusual opportunity was thus afforded all those in attendance, not only to observe the fine orchards and to gain a knowledge of the apple and peach industry in one of the most famous fruit growing districts of the eastern United States, but also to observe many important diseases of those crops, to study their control, and to discuss the problems involved with specialists in their own and in related fields.

A noteworthy feature of these pathological field meetings is the presence of foreign plant pathologists who are in attendance as the official representatives of their respective governments. Last year three well known potato pathologists attended the field meeting at Riverhead—A. D. Cotton of England, G. H. Pethybridge of Ireland, and H. M. Quanjer of Holland. This year the following official delegates from England, France, and Italy were present:—W. B. Brierly, Head of Department of Mycology, Institute of Plant Pathology, Rothamstead Experiment Station, Harpenden, England; Etienne Föex, Directeur de la Station de Pathologie vegetale, Paris, France; G. Rossati, Royal Agrarian Delegate to the Italian Chamber of Commerce, New York City; and K. Nakata, Professor of Plant Pathology, Agricultural Department, Kiushiu Imperial University, Fukuoka, Japan. The presence of these foreign pathologists not only greatly increases the interest and value of these conferences to their American colleagues, but also lays the foundation for international coöperation of inestimable importance to the progress of plant pathology.

The choice of locality for the 1920 phytopathological field meeting was especially fortunate. Agriculturally, the region visited is one of the most important of the United States. Scenically, the Shenandoah Valley and the Blue Ridge Mountains are famous throughout the world. Historically, every foot of ground traversed is of interest from Staunton on the south to Gettysburg on the north, and the party was able to make special pilgrimages to the battle-fields of Antietam and Gettysburg. And finally, the kindness of the people in the sections visited, their generous hospitality,

and their untiring efforts to make the phytopathological field meeting of 1920 both enjoyable and profitable, will remain a pleasant memory with all those who had the good fortune to be present.

G. R. LYMAN,
Chairman, Advisory Board of American Plant Pathologists

Stem-end rot of greenhouse tomatoes.—Late in January, 1920, the two southern commercial greenhouses at Macdonald College, Quebec, were planted with tomatoes. On May 2 a profuse growth of *Botrytis sp.* was observed on the stem end of a ripe tomato at the northwest end of the northern house. An examination of the surrounding plants revealed the fact that eight plants were affected. All showed grayish, slightly sunken areas on the stem similar to those described by Stone.[1] They were usually about a foot above ground, and varied in size from $\frac{1}{2}$ to 4 inches in length and from $\frac{1}{2}$ inch to a two-thirds girdling of the stem in width. Growth of the fungus on the stem was slow and sporulation weak in the greenhouse, but in a moist-chamber infected stems gave a profuse mycelial growth and sporulation was at a maximum. Cross sections of the diseased stems showed that the tracheae were discolored and in most cases the lumena were partially filled with a yellow-brown secretion. An interesting feature was the fact that the glandular trichomes characteristic of tomato and potato also showed this discoloration, while the epidermis and cortical tissues were apparently still normal. This was so marked that at a casual glance the stem would appear to be covered with minute sclerotia.

In addition to stem infection, by far the most infection occurred on the calyx, fruit stalk, and stem end of the fruit, and in these cases sporulation was much more pronounced in the greenhouse. As the fruit develops the sepals curl back on the pedicel, and an area about 2 mm. wide around the stem end of the fruit is exposed. It is in this area that infection of the fruit mostly occurs. Cook and Schwartze[2] point out that in blossom-end rot of greenhouse tomatoes caused by *Botrytis sp.* the disease was most common on the roughened blossom ends. Once the calyx is infected fruit infection rapidly follows, whether the fruit be green or mature. Sporulation is profuse (fig. 1) and as the mycelium advances it is preceded by a characteristic water-soaked area, which is well shown in figure 1.

The two houses were connected for ventilation purposes, air movement being maintained by the removal of areas of glass in the common wall

[1] Stone, R. E. A new stem-rot and wilt of tomatoes. Phytopathology **9**: 296–298. July, 1919.

[2] Cook, M. T., and Schwarze, C. A. Two interesting diseases of greenhouse tomatoes. Phytopathology **6**: 364–366. August, 1916.

of the two houses. The door communicating with the corridor was at the end of the greenhouse where infection was first observed, and the opening of this door at any time in the spring gave rise to a strong current of air through the two houses. There was at the same time an infestation of white fly (*Aleyrodes vaporariorum* Westw.). Without doubt the opening of the door caused hundreds of the flies to be carried throughout the houses and spores were distributed by these carriers. By May 15 the disease had spread through both houses, very few plants remaining free from infection.

When the fruit was attacked it would in most cases fall, unless it happened to be in the middle of a bunch, when it would be retained in place

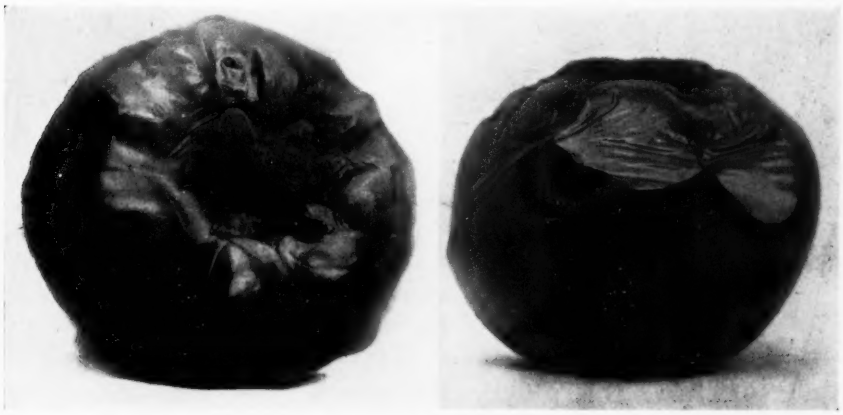

FIG. 1. STEM-END-ROT OF GREENHOUSE TOMATOES

Showing at the left profuse sporulation of Botrytis sp. on the stem end of tomato. The surrounding water-soaked area is also shown. The right hand fruit shows clearly the water-soaked area caused by the advancing mycelium. Both natural infections.

by the others. The other fruits in the bunch would become diseased later, in all probability through mycelial infection.

Pure cultures of the fungus were made on sterilized young tomato-stem plugs, on green tomato, on ripe tomato, on tomato leaves, and on glucose agar. In all cases there was luxuriant mycelial growth followed by abundant sporulation. At first the mycelium was grayish-white but as the culture aged it became grayish-brown. The spores were egg-shaped, cinereous, and large, being on an average 8μ by 12μ.

No sclerotia developed on material in the greenhouse, and in laboratory tests they developed in one case only. On May 3 a culture was made on a sterilized young green fruit, and by May 28 there were sixty-three small black sclerotia averaging 1 mm. in diameter.

No wilting of the plants occurred at Macdonald College, as no case was observed of complete girdling of the stem, but the fruit infection assumed serious proportions.

B. T. DICKSON

MACDONALD COLLEGE
QUEBEC, CANADA

Sclerotinia wilt of greenhouse tomatoes.—On the morning of April 26, 1920, my attention was called to a badly wilted tomato plant in one of the commercial greenhouses at Macdonald College, Quebec. According to the greenhouse attendant, the plant was apparently in good health on the evening of April 24.

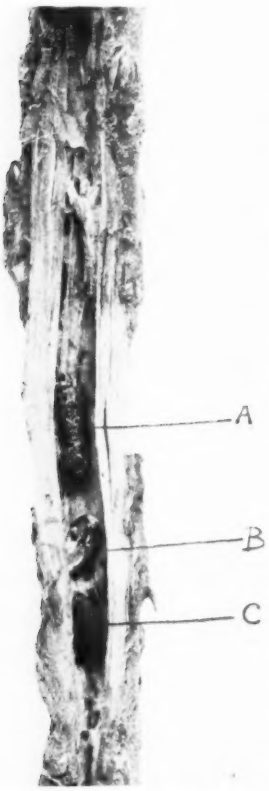

FIG. 2. SCLEROTINIA WILT OF TOMATO

Opened tomato stem showing three sclerotia of *Sclerotinia libertiana*.

On examination the base of the stem was found to have a slight covering of whitish mycelium for a distance of 6.5 cm. above ground. At this height above the soil level there was a slight injury where the first leaf had been removed, probably during cultivation.

Upon opening the stem three very fine black sclerotia were found, together with a little white mycelium, occupying the center in place of the pith. All the pith was gone up to a height of 10 cm. above soil level and, also, part of the woody tissues. The uppermost sclerotium was 7.5 cm. and the lowest 3 cm. above ground level. They measured respectively 3.5 mm. by 14 mm. (fig. 2, *A*), 3 mm. by 7 mm. (fig. 2, *B*), and 4.5 mm. by 8 mm. (fig. 2, *C*).

One sclerotium was washed in $HgCl_2$ solution and then in distilled water and planted in a test-tube containing sterilized green tomato fruit, on May 3. It was kept at room temperature, and by May 15 there was a profuse growth of white mycelium typical of *Sclerotinia libertiana* Fcl. On May 17 sclerotia to the number of sixteen had developed in the culture. They were similar to those which developed in the tomato stem, and were on an average 3.5 mm. by 7 mm. in measurement.

By May 8 the plant standing next to the one first discovered succumbed in a similar manner. Examination again revealed black sclerotia to the number of five, occupying the medulla of the diseased plant at a height of 16 cm. above soil level. On sterilized tomato plugs these gave rise to abundant white mycelium of *Sclerotinia libertiana*, and later numerous sclerotia developed.

The bed in which the tomatoes were growing had previously been planted with lettuce, which had suffered from an attack of lettuce "drop," and sclerotia had been collected from this part of the bed in December, 1919, on diseased lettuce leaves.

There was no yellowing of the leaves of the plant prior to the wilting, the wilt setting in quite suddenly.

This disease is apparently similar to that of potato known as "stalk disease," which is second only to late blight in importance in many areas of Ireland, and which is stated to be caused by *Sclerotinia sclerotiorum*.

B. T. DICKSON

MACDONALD COLLEGE
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A suspected mosaic disease of sweet clover. Mosaic is now of such wide distribution and economic importance, that its occurrence on a new host is worthy of note. A mosaic condition of sweet clover plants has, no doubt, attracted the notice of many; yet so far as can be learned, no mention has been made of it in literature dealing with plant diseases. In this note it is proposed simply to record its occurrence and the results of certain preliminary experiments carried out in the laboratory during the summer months.

The disease is without doubt quite common on sweet clover. Dr. Güssow states that throughout eastern Canada he has noticed the disease in a great number of localities. It was first seen by him in 1912 in plants in the vicinity of St. Catherines, Ontario. In the vicinity of Ottawa it is of very common occurrence. In almost every patch where the sweet clover was found growing wild, the disease was present. In most cases approximately half the plants were affected. In the plots of the Division of Agrostology on the Experimental Farm, where the plant is grown for certain tests, practically every plant was found to be diseased. It is interesting to note that these plots are the progeny of a limited number of parent plants. These parent plants are still living, and they also show distinct evidence of mosaic being present. It is not known, however, if the parent plants were affected at the time the seed was borne.

Affected plants show all the distinguishing symptoms that characterize Mosaic disease (fig. 3). The leaves have a mottled appearance and are

FIG. 3. SUSPECTED MOSAIC OF SWEET CLOVER, COMPARING PORTIONS OF DISEASED AND SOUND PLANTS. (SAME SCALE)

distorted and curled. The plants are somewhat dwarfed and exhibit an unhealthy appearance. When growing wild, diseased plants yield about one-half the herbage of their healthy neighbors. (In the cultivated plots the growth does not appear to be seriously interfered with.) Diseased and healthy plants are commonly found growing side by side. The disease is most apparent in new growth. In severe cases an early defoliation is noticeable.

During the summer months a number of inoculation experiments were made in the greenhouse. Plants for these experiments were grown in pots from ordinary commercial seed. All plants were free from mosaic. Juice was expressed from wild diseased plants and inoculations were made in the leaves by means of a sterile needle. On July 5 a set of twenty plants were thus inoculated; by July 27 twelve were distinctly diseased. On July 6 a set of fifteen plants were inoculated with juice used in former experiments after it had been passed through a Chamberland filter. Four of these plants became infected. Checks to these experiments remained healthy throughout the summer. Other inoculation experiments were made at later dates during summer. Some 140 plants were used in these, but there resulted only three infections. As the same procedure in making the inoculations was followed, the reason for the low percentage of infection is not known.

The writer has had the pleasure of discussing the results of the experiments with Dr. Güssow. He agrees that the disease is in every respect a typical mosaic. The inoculated plants develop the disease in the characteristic manner, i.e., by showing itself first in the new growth, and by not attacking fully formed leaves. He suggests that the particularly low percentage of infection in the later experiments was due probably to a higher temperature at the time of these inoculations, or perhaps to a change in the infectious qualities of the juice as the season advances.

Further experiments are being made, and a report will be made on these as results warrant. The writer wishes to express his appreciation of Dr. Güssow's interest and advice in these experiments.

H. R. MCLARTY

DIVISION OF BOTANY
EXPERIMENTAL FARMS

Plant pathology in the Philippines. The Department of Agriculture and Natural Resources of the Philippine government has recently organized a division of Mycology and Plant Pathology. H. Atherton Lee, formerly pathologist, Office of Fruit Disease Investigations, United States Department of Agriculture has been appointed Mycologist and Pathologist in charge. Mariano G. Medalla, formerly citrus canker agent, Bureau of

Plant Industry, United States Department of Agriculture, has been appointed Assistant Pathologist. Feliciano M. Clara, Felicisimo B. Serrano, and Gaudencio M. Reyes, graduates of the College of Agriculture, University of the Philippines, have been appointed scientific assistants. The laboratories of this newly created division have been established in the Philippine Bureau of Science and very complete laboratory equipment and a botanical library, unusually extensive for the Orient, have been made available for this work through the kindness of Director E. D. Merrill of the Bureau of Science.

Tropical phytopathology. The Tropical Research Laboratory of the United Fruit Company, which was formerly located at Zent, Costa Rica, and closed during the period of the war, is being reopened at Changuinola, Panama. Dr. John R. Johnston, Professor of Plant Pathology in the University of Havana, has been appointed Director of Tropical Research for the Company with headquarters in Havana, and two pathologists will be located at the Laboratory in Panama, one to continue work on the banana disease, and one on the diseases of the coconut, cacao and other crops.

Banana wilt in the Philippines. Plants of the Latundan variety of the banana, *Musa sapientum*, have been observed affected with banana wilt at Los Baños and Calamba in Laguna Province, and in several municipalities of Batangas Province, Philippine Islands. Affected plants show the typical yellowed leaves, sloughing off of leaf sheaths, split pseudostem, and reddish discolored rhizomes so excellently described by Brandes in Phytopathology, **9**, no. 9, September, 1919. A Fusarium species which agrees very closely with the description of *Fusarium cubense*, E. F. Smith, has been constantly isolated from the upper limits of discolored tissue in the pseudostem. This fungus upon inoculation with needle punctures into the pseudostem of healthy banana plants caused a blackening of the tissues which advanced rapidly longitudinally. Control punctures remained negative. Although the literature concerning the distribution of banana wilt is not all available here, we believe this to be the first observation of this disease in the Philippine Islands.

The disease is found in plantings eight to ten years old and being found in fairly well distributed localities is apparently of long duration in this country.

The abaca plant, *Musa textilis* Née, from which Manila hemp is obtained, is of course very closely related to the banana species. This plant is closely restricted in its distribution to the Phillippine Islands and its products constitute the most important items of export from this

country. For this reason we felt considerable concern as to the susceptibility of abaca plants to the banana wilt disease. Inoculations with *Fusarium cubense* upon abaca plants have in our preliminary experiments remained entirely negative, while inoculations from the same culture on banana plants have resulted positively. The evidence indicates that the abaca plant may be considered resistant to this disease.

H. ATHERTON LEE
FELICISIMO B. SERRANO

BUREAU OF SCIENCE
MANILA, P. I.

LITERATURE ON PLANT DISEASES[1]

COMPILED BY ALICE C. ATWOOD, AND FLORENCE P. SMITH, BIBLIOGRAPHICAL ASSISTANTS, BUREAU OF PLANT INDUSTRY

August, 1920

Adams, James Fowler. Rusts on conifers in Pennsylvania. Pennsylvania Agr. Expt. Sta. Bul. 160: 3–30, illus. 1919.

——— **Sexual fusions and development of the sexual organs in the Peridermiums.** Pennsylvania Agr. Expt. Sta. Bul. 160: 31–77, illus. 1919.

Literature cited, p. 66–67.

Anderson, Harry Warren. Dendrophoma leaf blight of strawberry. Illinois Agr. Exp. Sta. Bul. 229: 127–136, illus. 1920.

Due to fungus identical with *Phoma obscurans* E. & E., for which the author proposes new comb. *Dendrophoma obscurans*.

Ashby, S. F. Report of microbiologist, 1918–1919. Ann. Rpt. Dept. Agr. Jamaica **1918/19:** p. 26. 1919.

Banana diseases, Panama disease or banana wilt; Budrot of coconuts, one type of which is due to a fungus closely related to or identical with Pythium palmivorum Butler, the cause of budrot in India.

Association of Economic Biologists. The integration of mycological research with practice in agriculture, horticulture and forestry. Ann. Appl. Biol. **6:** 317–348. Ap. 1920.

Symposium held Dec. 11, 1919. i. The administrative problem, by A. D. Hall.—ii. The training problem, by V. H. Blackman.—iii. The agricultural problem, by E. J. Russell.—iv. The horticultural problem, by F. J. Chittenden.—v. The forestry problem, by W. Somerville.—vi. General discussion.

Atwood, George Gray. Report of bureau of plant industry. 1st Ann. Rpt. N. Y. Dept. Farms and Markets [**1917**]/**18:** 114–130. 1919.

Averna-Saccá, Rosario. Molestias do araçazeiro (Psidium araçá). Bol. Agr. [São Paulo] **21:** 43–45, illus. Ja./Mr. 1920.

Puccinia psidii, Wint. associated with anthracnose *Gloesporium fructus psidii*.

——— Molestias do cambucazeiro (Rubachia glomerata). Bol. Agr. [São Paulo] **21:** 37–42, illus. Ja./Mr. 1920.

Puccinia (*Uredo flavidula* Wint.), *jambulana* Rang.; anthracnose, *Gloesporium fructus*.

———. Molestias cryptogamicas do cacaueiro (Theobroma cacao) e do coquiro (Cocos nucifera). Bol. Agr. [São Paulo] **21:** 46–186, illus. Ja./Mr. 1920.

Literature in footnotes.

[1] This list includes references, both American and foreign, to the literature of plant pathology and mycology of interest to pathologists. Foreign references published since January 1, 1917, have been included beginning with the list appearing in v. 7, no. 3, June, 1917.

All authors are urged to cooperate in making the list complete by sending their separates and by making corrections and additions, and especially by calling attention to meritorious articles outside of regular journals. Reprints or correspondence should be addressed to Miss E. R. Oberly, Librarian, Bureau of Plant Industry, U. S. Dept. Agric., Washington, D. C.

Bal, S. N., and **Choudhury, H. P.** Commentationes mycologicae, 2–7. Jour. Dept. Sci. Univ. Calcutta **2** (Bot.): 1–9, 31–36. 1920.

2. Macrosporium (Fries) growing on *Citrus medica* (var. *acida*) and other species of Citrus.—3. Exoascus (Fückel) on *Nephelium litchi*.—4. Alternaria Nees, on leaves of (a) *Nicotiana plumbaginifolia*, and (b) *Datura stramonium*.—5. *Vermicularia jatrophae* Speg. on *Jatropha integerrima*.—6. *Phyllosticta glycosmidis*, Sydow and Butler, on *Glycosmis pentaphylla*, Corr.—7. A short study of *Plicaria repanda* (Wahl) Rehm. on *Borassus flabellifer*, Linn.

Ball, Elmer Darwin, and **Fenton, F. A.** Potato leaf hopper and the leafburn. Potato Mag. **2** (12): 6–7. illus. Je. 1920.

"It seems worth while to record the present state of our knowledge."

Beck, Olga. Über eine Methode der Saatgutuntersuchung auf Brand und über das Versagen der Kupfervitriolbeize. Naturw. Ztschr. Forst u. Landw. **18**: 83–99. Mr./Ap. 1920.

Literature in footnotes.

Biffen, Rowland Harry. Annual report for 1919 of the botanist [of the Royal agricultural society]. Jour. Roy. Agr. Soc. England **80**: 407–411. 1919.

Bourdot, H., and **Galzin, A.** Hyménomycètes de France. VI. Astérostromés. Bull. Soc. Mycol. France **36**: 43–47. Mr. 31, 1920.

Braun, Harry. Presoak method of seed treatment: a means of preventing seed injury due to chemical disinfectants and of increasing germicidal efficiency. Journ. Agr. Research **19**: 363–392, pl. 69–82. Jy. 15, 1920.

Literature cited, p. 392.

Brittlebank, Charles C. Flag smut (Urocystis tritici, Koern.). Jour. Dept. Agr. Victoria **18**: 240–243, illus. Ap. 1920.

Broili, Josef. Solanum edinense Berthault, ein für die Landwirtschaft Wertvoller Kartoffelbastard. Deut. Landw. Presse **47**: 359–360. Je. 23, 1920.

The *S. etuberosum* of Sutton, but not identical with *S. etuberosum* of Lindley. Very resistant to Phytophthora.

Brooks, Archibald Joseph. Efforts made to prevent the introduction of serious plant pests and diseases into Saint Lucia, and the spreading therein. Agr. Dept. St. Lucia, Leaflet 17, 12 p. D. 1919.

Büren, Gunther von. Beitrag zur Kenntnis des Mycels der Gattung Volkartia R. Maire (v. Büren). Mitt. Naturf. Gesell. Bern **1916**: 112–124, illus., col. pl. 1917.

Zitierte Literatur, p. 124.

Volkartia perennial in subterranean organs of host but appears to cause no substantial injury.

———. Untersuchungen über die Entwicklungsgeschichte und Biologie der Protomycetaceen. (Abstract.) Mitt. Naturf. Gesell. Bern **1916**: xlvii–l. 1917.

Burger, Owen Francis. Decay of fruit during process of marketing. Trans. Peninsula Hort. Soc. **33** (Delaware State Bd. Agr. Bul., v. 9, no. 3): 62–65. 1920.

Burkholder, Walter H. The effect of two soil temperatures on the yield and water relations of healthy and diseased bean plants. Ecology **1**: 113–123, illus. Ap. [Ag.] 1920.

California. Department of Agriculture. Horticultural statutes of the states of California. Corrected to November 1, 1919. 174 p., map. Sacramento, 1919.

Canada. Experimental farms. Division of botany. Mosaic disease in potatoes. Agr. Gaz. Canada **7**: 557–558. Jy. 1920.

Experiment to ascertain if potatoes from seed grown in disease free areas, can be grown in fields where the disease exists, without diminishing the first year's crop.

Chenantais, J. E. Sillon et pores germinatifs. Bul. Soc. Mycol. France **36**: 29–33. Mr. 21, 1920.

Chiovenda, Emilio. Nuova localita italiana per il Myriostoma coliforme (Dichs.) Corda. Nuov. Gior. Bot. Ital. n. s. **27**: 7–10. Ja. 1920.

Chipp, Thomas Ford. A host index of fungi of the Malay peninsula. II. Gard. Bul. Straits Settlem. **2**: 276–282. Je. 28, 1920.

Collins, James Franklin. Note on resistance of chestnut to the blight. Phytopathology **10**: 367–371, illus. Jy. 1920.

Cook, Melville Thurston. Lessons learned from seed inspection last season. New Jersey Dept. Agr. Bul. 24: 280–284. 1920.

Meeting New Jersey state potato association Ja. 15, 1920.

Cool, Catharina. Trametes pini (Brot.) Fr. nieuw voor Nederland. Nederl. Kruidk. Arch. **1919**: 126–128, illus. My. 31, 1920.

Dastur, Jehangir Fardunjii. Glomerella cingulata (Stoneman) Spauld. and v. Sch. and its conidial forms, Gloeosporium piperatum E. and E. and Colletotrichum nigrum E. and Hals on chillies and Carica papaya. Ann. Appl. Biol. **6**: 245–268, pl. x. Ap. 1920.

Literature in footnotes.

Gloesporium piperatum and *Colletotrichum nigrum* considered identical with *Glomerella cingulata*, and latter synonymous with *Gnomoniopsis* (*Glomerella*) *piperata* Stoneman. New disease of *Carica papaya* caused by conidial forms of a Glomerella identical with that on chillies.

Dietel, Paul. Über die Aecidiumform von Uromyces genistae tinctoriae. Ann. Mycol. **17**: 108–109. 1919 [1920].

Aecidium euphorbiae.

Doidge, Ethel Mary. The eradication of citrus canker. Jour. Dept. Agr. So. Africa **1**: 124–134, maps. My. 1920.

Act (No. 10, 1919) to amend the Agricultural pests act, 1911, in respect of land or plants infected with citrus canker, p. 130, 134.

Dox, Arthur Wayland, and **Roard, G. W., Jr.** The utilization of α methylglucoside by Aspergillus niger. Jour. Biol. Chem. **41**: 475–481. Ap. 1920.

Edgerton, Claude Wilbur. Albert Thomas Bell. Phytopathology **10**: 373–374, port. Jy. 1920.

——— and **Moreland, C. C.** Tomato wilt. Louisiana Agr. Expt. Sta. Bul. 174: 1–54, illus. Ap. 1920

Literature cited, p. 54.

Essig, Edward Oliver. New hosts of oak-root fungus in Humboldt. Mo. Bul. State Com. Hort. [California] **8**: 79–80. F. 1919.

Fischer, Eduard. Neuere Forschungen über Hausschwamm. (Abstract.) Mitt. Naturf. Gesell. Bern **1916**: vi. 1917.

———. Ueber eine Botrytis-Krankheit der Kakteen. Reprinted from Schweiz. Obst- u. Gartenbauztg. **22**: 106–107. 1920. 1 leaf.

Fitzpatrick, Harry Morton. Monograph of the Coryneliaceae. Mycologia **12**: 206–237, pl. 12–18. Jy. 1920.

Foëx, Étienne Edmond. Quelques causes de dégénérescence chez la pomme de terre. Jour. Soc. Nat. Hort. France IV, **21**: 204–207. Je. 1920.

Same article previously noted in Compt. Rend. Acad. Agr. France **6**: 398–404. 1920.

Francis, W. Orchard pests and how to control them. Journ. Agr. So. Aust. **21**: 951–954. Jy. 1918.

Fungus diseases: apricot scab, curl leaf of the peach, black spot of apple and pear.

Freeman, Edward Monroe. Division of plant pathology and botany. Minnesota Agr. Exp. Sta. 27th Ann. Rpt. **1918/19**: 54–56. 1919?

Fron, Georges, and **Lasnier, E.** Sur une chytridinée parasite de la luzerne. Bul. Soc. Mycol. France **36**: 53–61, illus., pl. 5. Mr. 31, 1920.

Bibliographie, p. 60.

Fulmek, Leopold, and **Stift, Anton.** Über in Jahre 1916 erschienene bemerkenswerte Mitteilungen auf dem Gebiete der tierischen und pflanzlichen Feinde der Kartoffelpflanze. Centralbl. Bakt. II, **51**: 97–129. Je. 10, 1920.

Gäumann, Ernst. Ein kleiner Beitrag zur Pilzflora des Krakatau. Bul. Jard. Bot. Buitenzorg III, **2**: 8–9, pl. 1. My. 1920.

Macrophoma arundinae n. sp. on *Arundina speciosa; Phyllostica tinisporae* n. sp. on *Tinispora crispa.*

Gauch, Achille, and **Durand, Joseph.** Quelques considérations sur la nature de la maladie du court-noué. Prog. Agr. et Vit. **74**: 504–543. Je. 6, 1920.

See also p. 536.

Gonzales Fragoso, Romualdo. Datos para la deuteromicetologia catalana. Mem. R. Acad. Cienc. y Artes, Barcelona III, **15** (17), 429–467. illus. My. 1920.

Gortner, Ross Aiken. Biochemistry of resistance to disease in plants. Minnesota Agr. Expt. Sta. 27th Ann. Rpt. **1918/19**: 34–35. 1919?

Great Britain. Ministry of Agriculture. Insect and fungus pests in July and August [1920]. Jour. Min. Agr. Gt. Brit. **27**: 375–378. 1920.

"It is proposed to include periodically in this Journal notes on insect and fungous pests which are likely to be troublesome in the month following."

Haack. Zur Kienzopf-Krankheit. Ein erneuter Infektionserfolg mit Äcidiensporen. Ztschr. Forst- u. Jagdw. **48**: 255–258. My. 1916.

Hall, Constant Johan Jacob van. Cacaokanker. Teysmannia **31**: 106–113. 1920.

Harvey, Rodney Beecher. Hydrogen ion changes in the mosaic disease of tobacco plants and their relation to catalase. Jour. Biol. Chem. **42**: 397–400. Jy. 1920.

Hedgcock, George Grant, Hunt, Nicholas Rex, and **Hahn, Glenn Gardner.** New species and relationships in the genus Coleosporium. Mycologia **12**: 182–198. Jy. 1920.

Publications cited, p. 197–198.

Hedrick, Ulysses Prentiss. Factors affecting hardiness in fruits. Massachusetts State Dept. Agr. Circ. 6, 10 p. Mr. 1919.

Symposium of experiences on varying behavior of peach trees during freezes and frosts.

Heinricher, Emil. Zur Biologie der Blüte von Arceuthobium. Naturw. Ztschr. Forst- u. Landw. **18**: 101–107. My./Ap. 1920.

Comment on Tubeuf's "Überblick über die Arten der Gattung Arceuthobium (Razoumowskia)" in v. 17 of same journal.

Heinricher, Emil. Zur Kenntniss der Verhältnisse zwischen Mistel und Birnbäumen. Ztschr. Pflanzenkrank. **30**: 41–51, illus. 1920.

Literature in footnotes.

Höhnel, Franz Xavier von. Mykologische fragmente. (CCXCI–CCXIV). Ann. Mycol. **17**: 114–133. 1919 [1920].

Houard, C., and **Lortet, M.** Des effets de l'hiver 1916–1917 sur les plantes du Jardin botanisque de Caen. Bul. Soc. Linn. Normandie VI, **10:** 184–212. 1919 [1920?]

Jardine, C. A. Eelworm in daffodils. Garden **84:** 374–375. Jy. 24, 1920. Continued from p. 361.

Keissler, Karl von. Ueber Pilze auf Orchideen im Reichenbachschen Herbar. Beih. Bot. Centbl. I, **36:** 307–319. 1918.

Killian, Karl. Über die Blattfleckenkrankheit der Tomate hervorgerufen durch Septoria lycopersici. Ztschr. Pflanzenkrank. **33:** 1–17, illus. 1920.

Verzeichnis der wichtigsten Literatur, p. 17.

Koeck, Gustav. Krankheiten und Schädlinge der Wurzeln und Knollen unserer gebräuchlichsten Ziergewächse. (conclusion) Ztschr. Gart. u. Obstb. F. 1, **1:** 39–40. Jy. 15, 1920.

Lemée, E. Notes de pathologie végétale. Bul. Soc. Linn. Normandie VI, **10:** 40–44. 1919 [1920?]

Puccinia rumicis f. *hydrolapathi* on *Rumex hydrolapathum; Erysiphe lamprocarpa* on *Stachys palustris; Erysiphe evonymi-japonici* on *Evonymus pulchellus; Calcoladia penicillata* (*Oidium erysiphoides* f. *viburnum-tini*) on *Viburbum tinus.*

Lüdi, Werner. Über die Zugehörigkeit der Aecidium petasitis Sydow. (Abstract.) Mitt. Naturf. Gesell. Bern **1916:** xxv. 1917.

Teleutospore form on *Festuca pulchella* belongs to Uromyces or Puccinia; author proposes the name *P. petasiti-pulchellae.*

Luyk, A. van. Über Gloeosporium tremulae (Lib.) Pass. und Gloeosporium populialbae Desm. Ann. Mycol. **17:** 110–113, illus. 1919 [1920].

Author finds these forms identical, but makes new genus, Titaeosporina (closely related to Titaeospora of Bubak) with one species: *T. Tremulae.*

MacMillan, Howard Gove, and **Byars, Luther Parris.** Heat injury to beans in Colorado. Phytopathology **10:** 365–367, illus. Jy. 1920.

Mann, Harold Hart, Nagpurkar, S. D., and **Kulkarni, G. S.** The "Tambera" disease of potato. Agr. Jour. India **16:** 282–288, pl. 15–16. My. 1920.

Caused by a mite of the group Tetranychidae.

Manns, Thomas Franklin. Report of fungus diseases for 1919. Trans. Peninsula Hort. Soc. **33** (Delaware State Bd. Agr. Bul., v. 9, no. 3): 79–84. 1920.

Martin, William Hope. The relation of sulfur to soil acidity and to the control of potato scab. Soil Sci. **9:** 393–408. Je. 1920.

References, p. 407–408.

Maublanc, André. Contribution à l'étude de la flore mycologique brésilienne. Bul. Soc. Mycol. France **36:** 33–43, pl. 294. Mr. 31, 1920.

——— and **Navel, H. C.** Sur une maladie du palmier à huile (Elaeis guineensis Jacq.) aux îles San-Thomé de Principe produite par un champignon (Ganoderma applanatum Pers.) Agron. Colon. **4:** 187–191, pl. My./Je. 1920.

Molliard, Marin. Tumeurs présentées par les racines du chou-rave et de la betterave. Bul Soc. Path. Vég. France **7:** 17–19. Ja./Mr. 1920.

Not due to Plasmodiophora; possibly akin to crown galls described by E. F. Smith.

Nedĕlchev, N. . . . . La brunissure de la vigne. Rev. Inst. Rech. Agr. Bulgarie **1:** 189–191. 1920.

Bulgarian text with French résumé.

Neger, Franz Wilhelm. Die Rolle des Lichts und des Chlorophylls bei der Entstehung von Rauchschäden an grünen Pflanzen. Ztschr. Forst-u. Jagdw. **48:** 624–635, illus. D. 1916.

Nowell, William. Mycologist's report on a visit to Trinidad. December, 1918–February, 1919. Proc. Agr. Soc. Trinidad and Tobago **19:** 141–159. 1919.

Passy, Pierre Félix. A propos de la filosité des pommes de terre. Jour. Soc. Nat. Hort. France IV, **15:** 500–501. Jy./D. 1914.

Peltier, George Leo, and **Frederich, W. J.** Relative susceptibility to citrus-canker of different species and hybrids of the genus Citrus, including the wild relatives. Jour. Agr. Research **19:** 339–362, pl. 57–68. Jy. 15, 1920.

Literature cited, p. 361–362.

Petrak, Franz. Mykologische Notizen. Ann. Mycol. **17:** 59–100. 1919 [1920].

Puttemans, Arsène. Sur l'Oidium du chêne au Brésil. Bul. Soc. Vég. Path. France **7:** 37–40. Ja./Mr. 1920.

Microsphaera quercina on oaks.

———. Une nouvelle maladie de la vigne—L'arrachement des grappillons. Bul. Soc. Path. Vég. France **7:** 34–36, illus. Ja./Mr. 1920.

Putterill, Victor Armsby. Flag smut of wheat. Jour. Dept. Agr. So. Africa **1:** 252–257, illus. Je. 1920.

———. A new apple tree canker. So. Afr. Jour. Sci. **17:** 258–271, illus., pl. 21–30. O./D. 1919.

References, p. 270.

Due to apparently undescribed fungus for which is proposed the name *Botryosphaeria* mali n. sp.

Rane, F. W. The chestnut bark disease and the chestnut market in Massachusetts. . . . . 30 p., illus., 4 pl. Boston, 1916.

Reed, George Matthew, and **Dungan, George H.** Flag smut and take-all. Illinois Agr. Expt. Sta. Circ. 242: 1–4, illus. Jy. 1920.

Reinking, Otto A. Higher Basidiomycetes from the Philippines and their hosts, II. Philippine Jour. Sci. **16:** 176–179. F. [Ag.] 1920.

Riddle, Lincoln Ware. Observations on the genus Acrospermum. Mycologia **12:** 175–181, pl. 11. Jy. 1920.

A. maxoni Farlow sp. nov.

Roberts, John William. The apple blotch and bitter-rot cankers. Phytopathology **10:** 353–357. Jy. 1920.

Rorer, James Birch. Black spot and bark rot of cacao. Agr. News [Barbados] **19:** 206. Je. 25, 1920.

"Freely translated from the Spanish of Mr. Rorer's handbook on the diseases of cacao in Ecuador."

Caused by Sphaeronema.

Rytz, Walther. Ueber Synchytrium, eine Gruppe einfachster, gallenerzeugender Pilze. (Abstract.) Mitt. Naturf. Gesell. Bern **1916,** p. 27–30. 1917.

Saccardo, P. A. Notae mycologicae, ser. XXIX. Micromycetes Dakotenses et Utahenses a Doct. J. F. Brenokle lecti et communicati. Mycologia **12:** 199–205. Jy. 1920.

Salmon, Ernest Stanley. On forms of the hop (Humulus lupulus L. and H. americanus Nutt.) resistant to mildew (Sphaerotheca humuli (DC.) Burr.) Ann. Appl. Biol. **6:** 293–310. Ap. 1920.

Bibliography, p. 210.

Schaffnit, Ernst. Versuche zur Bekämpfung des Kartoffelkrebses im Jahr 1918/19. Ztschr. Pflanzenkrank **30:** 59–67. 1920.

Schmitz, Henry. Shoe-string root rot of Rhododendron and Azalea caused by Armillaria mellea Vahl. Phytopathology **10:** 375, illus. Jy. 1920.

Schoene, W. J. Twelfth report of the State entomologist and plant pathologist. Quart. Bul. Virginia State Crop Pest. Com. **1**, no. 4, 28 p. Ja. 1920.

Inspection for pine blister rust, p. 14–15; Cedar rust and cedar eradication, p. 15–18; "Take-all" disease of wheat in Virginia, p. 22–23.

Shapovalov, Michael. The potato-scab control problem as it stands today. Potato Mag. **2** (11): 8–9, 26, illus. My. 1920.

Snell, Walter H. Observations on the distance of spread of aeciospores and Urediniospores of Cronartium ribicola. Phytopathology **10**: 358–364. Jy. 1920.

Literature cited, p. 364.

Spafford, W. J. Wheat and its diseases. Jour. Agr. So. Aust. **21**: 947–949. Jy. 1918.

Sydow, Hans. Ferdinand Theissen S. J. Ann. Mycol. **17**: 134–139. 1919 [1920].

Schriftenverzeichnis, p. 137–139.

Sydow, Hans, and **Sydow, Paul.** Über einige Uredineen mit quellbaren Membranen und erhöhter Keimporenzahl. Ann. Mycol. **17**: 101–107. 1919 [1920].

New genera of Pucciniaceae: Ctenoderma based on *Uredo cristata and toddaliae;* Dichlamys on Uromyces trollipi; Haplopyxis on *Uropyxis crotolariae;* Trochodium on *Uromyces ipomoeae.*

Tagg, Harry F. The preservation of artificial cultures of moulds. Trans. and Proc. Bot. Soc. Edinb. **27**: 335–337. 1919.

Traverso, Giovanni Battista. Pier Andrea Saccardo. Riv. Biol. **2**: 145–148, port. Ja./F. 1920.

———. Un po' di storia della organizzazione del servizio fitopatologico in Italia. Bol. Mens. R. Staz. Patol. Veg. Roma **1**: 7–16. Ja./F. 1920.

Tubeuf, Carl von. Rückinfektion mit Peridermium pini (Cronartium asclepiadeum) von der Schlanzenwurz auf die Kiefer. Naturw. Ztschr. Forst-u. Landw. **18**: 99–101. Mr./Ap. 1920.

Infection from Cronartium-stage on *Cynanchum vincetoxicum.*

U. S. Department of Agriculture, Bureau of Plant Industry. The white pine blister rust. What it is—where it grows—how it spreads. What you should do about it. [4] p., illus. Sept. 1919.

Circular issued in co-operation with Bureau of Plant Industry, U. S. Department of Agriculture by Minnesota State entomologist and state forester, and New Hampshire, Forestry dept.

———. **Plant Disease Survey.** Plant disease bulletin. 4: 24–39, Ag. 1; 40–53, Ag. 15, 1920.

Valleau, William Dorney. Seed corn infection with Fusarium moniliforme and its relation to the root and stalk rots. Kentucky Agr. Expt. Sta. Bul. 226: 25–51, illus. 1920.

Literature cited, p. 51.

Research bulletin.

Van der Bijl, Paul Andries. The systematic position of the fungus causing root-disease of sugar-cane in Natal and Zululand. So. Afr. Jour. Sci. **16**: 204–206. O./D. 1919.

Himantia stellifera, not *Marasmius sacchari,* cause of root disease in Natal and Zululand.

Weir, James Robert. Note on the pathological effects of blazing trees. Phytopathology **10**: 371–373. Jy. 1920.

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